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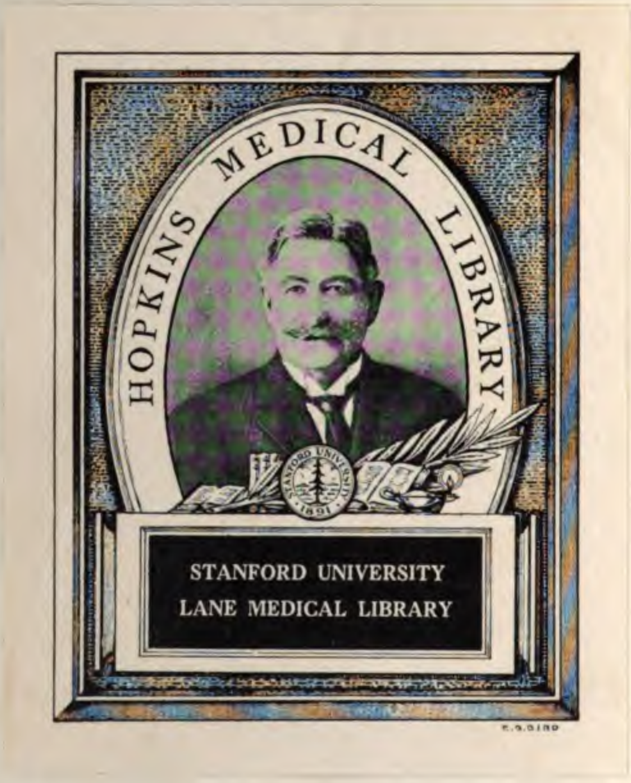

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A MANUAL

OF

PATHOLOGICAL ANATOMY.

BY

CARL ROKITANSKY, M.D.,
CURATOR OF THE IMPERIAL PATHOLOGICAL MUSEUM, AND PROFESSOR AT THE
UNIVERSITY OF VIENNA, ETC.

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TRANSLATED FROM THE LAST GERMAN EDITION

BY

WILLIAM EDWARD SWAINE, M.D.,
EDWARD SIEVEKING, M.D.,
CHARLES HEWITT MOORE,
GEORGE E. DAY, M.D., F.R.S.

FOUR VOLUMES IN TWO.

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VOLUME III.

THE

BONES, CARTILAGES, MUSCLES, AND SKIN,

CELLULAR AND FIBROUS TISSUE,

SEROUS AND MUCOUS MEMBRANE,

AND THE

NERVOUS SYSTEM.

TRANSLATED FROM THE GERMAN,

BY

CHARLES HEWITT MOORE,

SURGEON TO THE MIDDLESEX HOSPITAL;
LECTURER ON ANATOMY IN THE MIDDLESEX HOSPITAL SCHOOL OF MEDICINE.

PHILADELPHIA:
BLANCHARD & LEA.
1855.

yet more highly estimated in this country the more it is know Were there no other proof of this, it might be found in the needl labor that is still incurred by some English pathologists to arrive facts and opinions which have been already ascertained, and alrea weighed, by Rokitansky. From his work, as from a fresh starti point, such laborers may advance to new discoveries, without risk of having priority claimed by a foreign observer.

I have made an effort, with difficulty indeed, to avoid the in duction of new scientific terms, and have therefore ventured adopt a different title for the chapters from that which has b employed in the previous volume. I have done so, however, the less reluctance, as either mode of translating the original "Abnormitäten" is perfectly intelligible, and as none of its gen import appears to be lost, though divided between the two w "Anomalies and Diseases."

As to the other characters of the present volume, a translator best fulfilled his duty, if he seem not to intervene between author and his readers.

In the descriptions of microscopic appearances, I have m sought the judgment of my colleague, Mr. De Morgan. To and to my kinsman, Dr. Moore, I am most happy to offer my gra acknowledgment of their kind assistance in this, and not on this, professional effort.

C. H.

MORTIMER STREET, CAVENDISH SQUARE,
October, 1850.

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[Though the original of the line at p. 107, to which the foot-note refers, may be translated as in the text, its signification seemed determined by a more complete description of apparently the same growths at p. 167, to be "straight or convoluted elevations" *of* the surface, not exostoses separate from it.—Ed.]

PART IV.

ANOMALIES AND DISEASES OF CELLULAR TISSUE.

PART IV.

ANOMALIES AND DISEASES OF CELLULAR TISSUE.

§ 1. *Varieties in regard to quantity.*—The cellular tissue contained in the human body is subject to variations in quantity which come within the sphere of Pathology.

In some bodies this tissue is over-abundant, in others its quantity is smaller than usual; neither condition, however, constitutes more than an individual peculiarity.

But it may accumulate in excessive quantity in particular parts of the body, entering into the composition of various morbid growths and tumors, or forming a uniting medium between organs which in their natural state are separate from one another. As an areolar callus it may supply losses of substance which are otherwise irreparable, filling up cavities in the injured tissues; or it may become accumulated in excess, i. e. hypertrophied, at parts which have been subjected to continued irritation, as, for instance, in the neighborhood of inflamed spots. And, lastly, it may occupy the place of parts which, in consequence of some fault in their original formation, are wanting; such as the bulb, the rectum, or the thoracic organs in cases of acephalus; the muscles, and even the bones, of incompletely developed limbs, &c.

Nevertheless, the presence of a mass of cellular tissue in the spot once occupied by some previously existing organ, cannot always be regarded as an exuberant growth of that tissue; for that which remains behind after organs have disappeared, whether from primary or secondary atrophy, or in the ordinary course of natural decay (Involution), is nothing more than the tissue which once formed the connecting medium in their anatomical composition, and which, therefore, then occupied the same place. This is the case with the thymus gland, and the mammæ, with absorbent glands, with the ovaries, &c.

Exhaustive diseases produce general diminution of the quantity of cellular tissue; and thè same result ensues in particular portions of it, as well as in other organs, from continued pressure and from paralysis, and after suppuration and sloughing.

§ 2. *Anomalies in regard to Texture.*

1. *Hyperæmia, apoplexy of cellular tissue.*—Under suitable circumstances, particular portions of the entire system of cellular tissue are subjected to transient, or to permanent, congestions of an active, a passive, or a mechanical nature. These congestions, especially the two last-mentioned kinds, sometimes occasion spontaneous hemorrhages into the cellular tissue (apoplexia textus cellulosi); the effusions of blood are

serious character in particular localities; as when it attacks the cellular tissue of the trunk or limbs; or that between the pericranium and the galea aponeurotica of the head; that near the submaxillary (Ludwig), or the thyroid glands; or that which accompanies the trachea, pharynx, and œsophagus down to the mediastinum; or the tissue upon the lumbar vertebræ, or on the iliacus muscle; or that in the neighborhood of the cæcum (perityphlitis), or of the rectum (periproctitis), or bladder (pericystitis); as well as interstitial cellular strata, especially the submucous tissue of the stomach, intestinal canal, &c.

The inflammation, whether primary or secondary, may have a more or less distinctly marked exudative character; as is illustrated by phlegmatia alba, as it is called, amongst the primary inflammations, and by the numerous instances of inflammation which occur in cellular tissue by metastasis, after acute exanthemata, typhus, &c., amongst the secondary. They are often associated with exudative processes on membranous expansions, especially such as adjoin mucous and serous membranes.

Moreover inflammation of cellular tissue is frequently derived from that of other parts. It uniformly accompanies the inflammation of structures which are imbedded in it; especially inflammation of veins, and lymphatic vessels, lymphatic glands, nerves, muscles, serous and fibrous membranes, &c. It is generally subordinate in degree to that which prevails in the actual seat of disease; though there are occasional exceptions to this rule, the inflammation in the neighborhood of the actual seat of disease sometimes going on at isolated points to the more advanced degree, particularly to suppuration. Such is the case, for instance, in inflammation of veins.

Inflammation of the adipose tissue may subside, occasioning merely some loss of the fat: in process of time, however, the loss is supplied again.

But in other cases, severe inflammation is followed by coalescence and obliteration of the fat vesicles, and consequently by a marked shrinking of the part which has been inflamed.

Inflammation of this kind often terminates in suppuration; and the adipose tissue becomes converted into a yellowish-red pulpy mass, which is infiltered with a fatty purulent fluid.

Lastly, a fibrinous inflammatory product is sometimes seen in the fat vesicles; it solidifies in them, changes into a cheesy mass, and in the end even becomes cretaceous. The wall of the vesicle is then found thickened, it contracts upon its contents, and bears traces of its previous congestion in the slate-gray or blackish-blue color with which it is tinged. This change is principally observed on the fatty tissue of the omenta, and appendices of epiploacæ; especially in tubercular subjects, in whom peritoneal inflammations have occurred and have been attended with tubercular exudation.

Chronic inflammation of cellular tissue is distinguished by the following characters: the tissue is but slightly injected and reddened; though where it is exposed, as it is at the base of the ulcers, its color is a deep red, tinged with various shades of brown, coppery, rusty yellow, violet, &c.: it is also denser than natural, and contains a viscid serous fluid, which when ulcers exist, filters through to their surface.

Inflammation of this kind does not proceed to suppuration, except perhaps at a few small isolated spots: it tends rather to induration and that at an early period. The tissue is then very compact, and so tough that it makes a creaking noise when it is cut: it is also pervaded, and as it were identified, with a gelatinous matter, or an albuminous substance resembling lard. The tissue is found in these various conditions beneath and around chronic ulcers, in the neighborhood of fistulæ, after repeated and habitual, or mismanaged attacks of erysipelas, or as a consequence of mechanical hyperæmia, or congestions arising from varicose veins in the lower limbs, in cases of elephantiasis, &c.

An inflammatory process of a peculiar kind occurs in new-born children, and is known by the name of induration of the cellular tissue (sclerosis telæ cellulosæ). It occurs on the trunk, especially on the whole of the lower part of the abdomen, as well as on the thighs, and the cheeks. The subcutaneous fatty and cellular structures are moistened by a yellowish viscid serum, the fat is condensed and forms a yellowish or brownish-red hard granular mass, and the skin covering it is tense, as firm and resisting as a board, glistening and pale or of a yellowish-red color. The numerous theories which have been broached as to the nature of this induration of cellular tissue and its etiological relation with disorders of internal organs are daily proved to be unsound. Thus it has been traced to diseases of the liver or lungs, to permanent patency of the fœtal canals and cyanosis, and to irritation of the stomach and intestinal canal. But these conditions, and even the icterus, which is its most frequent associate, are all accidental complications. The induration is unquestionably an independent disease (occasioned by disorder of the functions of the skin of the new-born child), and, just as in burns of the integuments, its seriousness and danger are directly proportioned to its extent.

3. *Depositions, metastases*, are very frequent both in the subcutaneous and in the deeper layers of cellular tissue. The deposits are of purulent and ichorous nature, and are often very numerous and extensive. They sometimes result from a primary and spontaneous pyæmia, which has been occasioned by pus or sanies having been taken into the mass of the blood; and sometimes from a state of the circulating fluid, in which other processes, exanthemata, typhus, &c., have led to the generation of pus by the blood in a secondary manner.

4. *Gangrene of cellular tissue.*—Mortification is liable to take place in cellular tissue, not only as a consequence of inflammation, but under other conditions also, as a primary disease. Sometimes the tissue, at first congested, and dark red in color, changes into a blackish, very moist, shreddy, and friable pulp: at other times, after having formed a blackish-red, viscid pulp, it becomes a dry, tinder-like, crumbling eschar. Lastly, it sometimes degenerates into a *white* mass, shaded with dirty yellow or greenish, and is moist and extremely easily torn.

5. *Adventitious growths.*—The adipose and cellular tissue beneath the skin, as well as that which is collected in larger quantity in the internal regions of the body, is occasionally the seat of *cysts.* The contents of these growths are exceedingly various; sometimes being serous, sometimes resembling synovia, or gum (colloid), fatty, cholesteatomatous, or melanotic. *Fibrous tumors* occur in the same structures; *calcareous*

concretions are very seldom met with, the only instances being that in which a fibrinous exudation in the fat vesicles becomes, as has been mentioned, converted into chalk, and that of cord-like growths, or smooth, or tuberous plates of bone, which occur in the fibroid callus of which the cicatrix of cellular tissue is composed. *Tuberculous* matter is deposited in young persons usually, and especially in children; the depositions occur in the subcutaneous tissue, and are more or less circumscribed: they soften and form a cheese-like or fatty pulp, and then exciting an inflammatory process in the integuments, which ends in ulceration, they make their way outwards. They are always associated with tuberculosis of the lymphatic glands, and frequently with the same disease in other parenchymatous organs.

Sarcomatous and *cancerous* growths are frequent in cellular tissue, and of the latter it is the genuine white medullary form, and the cancer melanodes that chiefly occur.

Among the *entozoa*, the filaria medinensis is met with in the subcutaneous cellular tissue.

§ 3. *Anomalies of Secretion and accumulations of Foreign Bodies in Cellular Tissue.*—In the first place, the fat is subject to considerable deviations from its natural quantity and quality.

Not unfrequently it is found collected in excessive amount, and at the expense of the nutrition of other structures, especially of the muscles. The excess, when uniform throughout the body, constitutes what is expressed by the general term obesity, and to it the female sex is particularly liable: but in some cases it accumulates at particular spots externally, and disfigures the body; or internally in such a manner as to narrow the space of the cavities of the body, and to interfere with the functions of the organs contained within them. Thus it collects at the lower part of the abdomen, on the nates, and on the loins (in which region the rolls of fat are situated, which distinguish the race of Hottentot women); it is found, too, in the neighborhood and in the cellular interspaces between the lobes of the mammæ in women; in the mediastina, and beneath the pleura; in the folds of the peritoneum; around, and in the duplicatures of, synovial membranes, &c. The local accumulations just mentioned, constitute a sort of transition to the fatty tumor, *lipoma*. Invested with a cellular sheath, lobulated by interstitial cellular tissue, more or less of which traverses its interior, and resembling adipose tissue in its intimate texture, the fatty tumor chiefly occurs in those regions at which fat is naturally most abundant: it is, however, sometimes found in parts where in the normal state no fat is deposited; as, for instance, in the submucous cellular stratum of the intestinal tract.

Yet more frequently, in the emaciation which attends disease, the quantity of fat existing is found remarkably small, and at certain parts of the body, if not everywhere, it may even entirely disappear.

Moreover, the fat presents various qualitative deviations from its physical, and no doubt also from its chemical, properties. Sometimes it is remarkably pale, and sometimes, on the contrary, of a very dark color; it may be soft, gelatinous, suety, greasy, like the marrow of bones, and oily; or again, firm, and resembling soap or adipocire. Thus in advanced age, and in persons whose muscular system loses energy and bulk, and becomes prematurely aged, the fat is of a deep yellow color and oily; in spirit-drinkers, in persons who are negligent as to the state of their skin, in those whose skin is thick, soft, and dark colored, or in whom the liver is suety, or the heart the seat of fatty metamorphosis, &c., it is usually pale, and resembles mutton suet.

In dropsical patients it is often reddish, firm, and granular, the fat vesicles shrinking together, and forming a reddish firm acinus: but otherwise it disappears, and after it has been absorbed, its place is occupied by a fatty, gelatinous, and, at last by a serous, fluid.

The serum, which in the natural state, is uniformly diffused through the cellular tissue and moistens it, is subject to similar deviations from the healthy condition.

It is almost entirely wanting in cases of considerable general or partial emaciation, in the marked collapse which succeeds convulsive diseases, or when, as in serous diarrhœas and Asiatic cholera, the serum of the blood is rapidly lost, &c. In these cases the cellular tissue is dry and crepitant, and resembles that of plants. In other instances, again, it exceeds the natural quantity: this excess, when general, constitutes leucophlegmasia, hydrops universalis, anasarca; when local it is named œdema. The fluid varies much in color, consistence and composition, according to the processes by which its accumulation has been effected, as well as according to the composition of the blood; being either thin and clear as water; or rather thick, and like jelly, from containing albumen; or yellow, in consequence of the presence of bile; red, from the admixture of more or less blood with it; or turbid, milky, whey-like, and flocculent, from its containing fibrin or purulent matter.

Among the foreign bodies found in cellular tissue are:

a. Gas; which may be either atmospheric air or some of the various animal gases—*windgeschwulst*, emphysema. Atmospheric air accumulates in cellular tissue in consequence of wounds in the circumference of the thorax by which the pleura is opened; and more frequently it succeeds penetrating wounds of the lung, fractures of the ribs by which the costal pleura is torn and the lung injured, ruptures of the lung and pleura occasioned by crushing, mortification of the lung and superjacent pleura, ulcers which perforate the larynx and trachea, and laceration of one or more of the air-cells of the lung (from violent coughing, &c.) The emphysema is occasioned either immediately by the entrance of the atmospheric air at the wound of the chest, or by its escape from the air-passages into the adjoining tissue, or into the cellular structure which intersects the lung itself—emphysema pulmonum interlobulare. When the bowel is perforated either by ulceration or sloughing, its gaseous contents pass out into the cellular tissue: in some cases of mortification gas is spontaneously evolved, and the skin over it swells up, and forms a doughy tumor. Lastly, there are a few cases in which, without any of the above-mentioned causes, gas accumulates in the subcutaneous tissue, and still more frequently in the interstitial, and especially the submucous, cellular tissue of the bowel: such cases result from acute disorganizations of the blood, and are found when there is no trace of cadaveric decomposition in the body. Transient emphysemas of the same kind are well known to occur in the living subject, in consequence of convulsive affections.

PART V.

ANOMALIES AND DISEASES OF SEROUS AND SYNOVIAL MEMBRANES IN GENERAL.

§ 1. *Deficiency and Excess of Development in the System of Serous Membranes.*—There are various kinds of primordial defect in serous membranes. They may be entirely wanting. Sometimes the organs they should enclose are wanting too; but if these exist, the deficiency of the membrane is supplied by some adjoining serous expansion. They may be but partially developed. This is the case when serous cavities are fissured, or when, in consequence of a partial defect in the wall of separation between two serous sacs, their cavities have an unnatural communication with one another. Thus the cavity of the pericardium is sometimes continuous with that of the pleuræ, the peritoneum with the latter or with the tunica vaginalis of the testicle, bursæ communicate with the synovial cavities of joints, &c. Moreover, a deficiency in the development of a serous or synovial membrane may depend upon the absence of the organ usually contained within it; in which case, particular duplicatures of the membrane do not exist.

Wasting of a serous membrane, obliteration of its cavity, degeneration of it into cellular tissue (Rückbildung), and its destruction by suppuration, constitute instances of acquired defect.

Excess of development may present itself as a congenital anomaly. It then sometimes assumes the form of unusual saccular prolongations and duplicatures of the membrane, or of folds which, though such as usually exist, are preternaturally developed,—a form of excess which may partly be traced to an arrest of development: sometimes appearing under another form, as a serous cyst, or an aggregation of serous cysts, or as a honeycombed serous tissue, it marks the site of some originally defective part, or of an organ which had been diseased and destroyed during fœtal life,—the brain, for instance, in hemicephalus, or the kidneys.

It more frequently happens, that new serous tissue is formed at some period subsequent to birth; it may present the characters of true serous membrane, or may be cellulo-serous or fibro-serous. Thus,

a. Bursæ, which are the simplest form of the synovial system, are developed beneath the skin, where it is exposed to unusual and permanent pressure and friction, as is the case in clubfoot (Béclard), over the point of an angular curvature of the spine (Brodie), or on the stump of an amputated limb; and they are also observed in deeper parts, between muscles and tendons, as well as between them and the unusual protuberances of bone, &c. Moreover, dislocated bones are sometimes firmly fixed by tion of the congestion and the constitution of the blood, and varies from a bright to quite a dark color. The extent and the intensity of the redness and injection also differ widely in different cases; sometimes they are partial and scarcely perceptible, sometimes they are universal and stain the membrane through. In many serous membranes, particularly those which, like synovial membranes, or still more the arachnoid, have a very delicate structure, the reddening which takes place is usually very slight, or there may be none whatever: these membranes admit of injection with blood only with much difficulty, and the degree of redness and injection is no criterion of the intensity of the process, so far as regards the quantity of product which the inflammation will supply; for the exudation, especially in inflammations of a croupy character, is remarkably disproportioned to the redness and injection of the membrane.

β. *Opacity and thickening.*—In those parts where the membrane is reddened and injected, and still more evidently in the interspaces between them, it becomes dull, loses its lustre, transparency, and smoothness, and acquires opacity and a velvety internal surface. Serous membranes of very delicate structure, such as the arachnoid, become at once opaque, dull, and turbid, like whey or milk. This change, and to some extent also the thickening, are due to infiltration of the membrane; but the thickening is produced rather by the simultaneous affection of the subserous cellular tissue; for that tissue is always injected, is early filled with an opaque serous fluid, and is consequently tumid. The infiltration of the subserous tissue extends to the membrane itself, and the two become so blended as to lose all trace of separation from one another. From the expansion, or loosening, of the tissues which takes place, the serous membrane is rendered not only very fragile in itself, but also easily separable from the structures beneath it.

γ. The *effusion* or inflammatory product upon the free surface of the serous membrane is sometimes an exudation of plastic nature, but at other times, and especially in inflammations which arise from metastasis, it is diffluent and puriform, or actually purulent, or sanious. In quantity it is usually not very considerable, at least when compared with the amount of effusion attending chronic inflammation, though there are exceptions to this rule, especially in the case of exudations of a croupy character.

The plastic exudation is mostly accompanied by an effusion of serous fluid, so that the whole product may be distinguished into a plastic or coagulable portion, and another which is not coagulable. The relative quantity of the two portions varies considerably: there are some exudations which have no serous part whatever, while others contain no more of the plastic matter than suffices to render the serous effusion slightly opaque. An effusion of perfectly clear serous fluid, unaccompanied by any deposit of lymph upon the inner surface of the serous membrane, is scarcely such as can be attributed to actual inflammation.

The plastic portion of the exudation is deposited upon the inner surface of the serous membrane, and forms there a peripheral fibrinous layer which encloses the serous effusion, if any exist: it is of a grayish-red, a yellow, or a grayish color, and may vary in thickness from that of a scarcely perceptible film, like hoar frost, to that of several lines. Its inner free surface is sometimes tolerably smooth, sometimes villous, sometimes shreddy, sometimes areolar; occasionally it resembles waves of sand, or the back of a bullock's tongue. When the plastic matter is very abundant, it forms other large masses also of loose texture, and soaked through with more or less of the serum: it may also render the serum opaque, or may lie in it in flakes, which soon become arranged in plates and cords, and form a network or honeycombed cellular structure, the large interspaces of which enclose part of the serum. It may also fall upon the inner surface of the original peripheral coagulum, and form a soft shreddy covering for it; and in that case, the exudation lining the serous membrane consists of two layers:—the original, more consistent, plastic exudation; and the secondary, looser, shreddy precipitate upon it.

Inflammations of an eminently exudative character are particularly remarkable—

For the inconsiderable reddening and injection of the serous membrane:

For the disproportion which subsists between the reddening and injection, and the great quantity of exudation deposited at one time:

For the marked loosening of tissue, and infiltration observable both in the membrane and in the subserous structure:

For their frequently coexisting with exudative processes in mucous membranes, the plastic character of which may differ from that of the inflammation in the serous membrane:

And as a general rule also for the homogeneous nature of the whole product, and for the absence or mere indication of a separation of it into one part which is plastic, and another incapable of coagulation: it is a uniform exudation, which coagulates more or less, or degenerates into pus, or is sanious.

There is sufficient general connection between the exudations under consideration and another found upon large serous membranes, like the peritoneum and pleuræ, to allow of the latter being mentioned here. It is a viscid coating upon these membranes which gives them a dull lustreless aspect: it is best marked and most constantly seen in cases of Asiatic cholera, but it occurs also in the course of other exudative processes which are attended by exhaustion, such, for instance, as the diarrhœa of children.

Plastic exudations very frequently produce adhesions between the walls of a serous cavity and the viscera it contains, as well as of the viscera with one another: but a large quantity of serous effusion holds the lamellæ of the plastic exudation apart, and no adhesion can take place unless the fluid be absorbed before the lymph is completely organized.

Absorption commences as soon as the lymph is deposited, and the intensity of the inflammation subsides: it depends, therefore, upon the cessation of the inflammatory process; but it is also influenced by the thickness and density, that is to say, by the permeability, as well as by the stage of organization, of the lymph.

In the organization of plastic exudations, new vessels are spontaneously formed in more or less abundance, and a tissue is produced, which is either cellular, loose, and cellulo-serous; or of closer texture, strong and, as it is called, cellulo-fibrous. If the serous surfaces have been agglutinated together by the exudation, their complete vital adhesion is polished like gypsum, or rough and sandy, like mortar. Or lastly, the pseudo-membranous sac contains a fatty glutinous fluid, which is, for the most part, of a brownish-yellow color, and is mixed with numerous minute glittering scales (fat-crystals), which cling in thick clusters to its inner surface.

In the most unfavorable case, the purulent exudation becomes sanious, and for the most part, of a greenish color; at the same time, in some instances, gas is evolved, and the exudation assumes a most offensive pungent odor, like that of garlic, phosphate of ammonia, or sulphuretted hydrogen. Under these circumstances, the serous membrane not unfrequently ulcerates, or sloughs at some small and circumscribed, or at larger spots; sometimes the ulceration extends into the subserous structure, and opening of the serous sac takes place, and its contents are spontaneously discharged, either externally or into another cavity.—(Phthisis membranæ serosæ ulcerosa.)

γ. Chronic inflammation may extend from the serous membrane to the pseudo-membranous structure which lines it, and lead to a deposition of its products both within the substance, and on the internal free surface of the new structure; that is to say, the exudation deposited by one inflammatory process may itself become the site of a new inflammation during the time that it is becoming, but is not yet completely, organized. This explains the otherwise unintelligible occurrence of exudations upon the inner surface of serous membranes, which have already been converted into thick fibroid rinds, while effusions, to all appearance quite recent, have taken place into the cavity.

This secondary inflammatory process, occurring during the progress of organization in the plastic product of a previous acute, or even of a chronic, inflammation, deposits exudation both upon the free surface of that product, and also within its tissue or parenchyma: the former constitutes a second free exudation, the latter an infiltration. The infiltered product, during its organization, becomes an integral part of the original exudation, and renders it thick and very dense, compact in its parenchyma, and of fibroid or fibro-cartiliginiform structure: the stratum thus formed becomes identified with the serous membrane.

The free exudation may be, and usually is, a plastic one, with more or less also of an aplastic portion; it may, however, present any of the qualities, and undergo any of the metamorphoses, already detailed: indeed, the inflammation within the parenchyma of the exudation may even have a suppurative tendency, and abscesses may be formed in it, analogous to those which occur in the subserous cellular tissue. The new plastic exudation becomes organized into a cellular tissue, and unites with the older layer of exudation beneath it; and it again may be attacked by inflammation, and become callous and indurated.

In this manner tough, fibroid laminæ are formed upon serous membranes, which, if the process be repeated often enough, may measure three or six lines, and even an inch and more in thickness; they are united externally with the serous membrane, to which they become firmly and immovably fixed, or even with adjoining fibrous expansions, aponeuroses, periosteum, &c.

If no further inflammatory process take place in the last exuded layer, that layer becomes organized to cellular tissue; and if none of the effused fluid remain at the termination of the process, and the opposed layers come into contact with each other, they unite into a single layer, and the serous cavity is obliterated in whole or in part, according to the extent of the process. But if any of the earlier, or of the more recent, fluid effusion be still left, it is either kept encysted by the impermeability of the fibroid stratum, and the slight power of absorption which it possesses, or else it is slowly diminished in quantity by absorption. Whilst the serum is gradually being absorbed, the plastic (albuminous and gelatinous) matters which it contains are precipitated upon the walls of the fibroid exudation, and form a loose villous lining for it; or else they accumulate in one shapeless mass, and become encysted in some part, generally the most dependent part of the cavity. If under such circumstances, the walls of the serous cavity approximate and fall together, the layers of the exudation will be agglutinated to one another by means of a stratum of grayish jelly-like substance, into which the precipitate just described changes, and which Laennec, from his observations of it in pleuritic exudations compares to the central part of the intervertebral substances. In the end it becomes converted into a dense compact cellular tissue.

This process is mostly observed upon the pleura, and will be more particularly referred to amongst the diseases of that membrane.

An exudation of a peculiar nature is very frequently found upon serous membranes, and requires particular notice. Its true character was first discovered by Laennec, who named it the hemorrhagic exudation. It is usually large in quantity, and consists of fibrin, blood-corpuscles, coloring matter of the blood, and serum mixed in different proportions, and it is chiefly distinguished by its more or less intense red color.

The conditions out of which it arises are general and local. Those of the former kind include diseased states of the blood, particularly tuberculosis; the anomaly in the composition of the blood, which results from cirrhosis of the liver; the scorbutic constitution; and, that which is allied to the last two, the dyscrasia of drunkards. Besides these, there are, of course, red and variously discolored exudations, which proceed from decompositions of the blood, such as succeed exanthemata (variola and scartina), typhus, &c.

The following are the local conditions of its occurrence: The hemorrhagic exudation, though it sometimes, no doubt, results from primary inflammation of a serous membrane, yet it is far more frequently the product of the third mentioned form of chronic inflammation, i. e. of a secondary inflammatory process occurring in a plastic exudation; and this is, in fact, the cardinal local condition under which it takes place. For the structure in which the inflammation occurs is in course of organization; its vessels are only just forming, and have as yet no actual coats, or, at any rate, but very delicate and permeable ones; and they have not yet united into a freely inosculating circulatory system; from such a structure the exudation occurs, without question, repeatedly and at intervals, and probably also prematurely before the congestion has reached the degree of intensity which would, in any other structure, be necessary to produce it. The whole process bears throughout it the stamp of an inflammation which has not arrived at maturity: and its product is blood, altered by congestion in the composition, and mutual relation and intermixture of its elements. The fibrin, blood-corpuscles, and coloring matter contained in it vary with the state of the constitution, and with the composition, but more particularly with the stage of organization, of the substratum itself. It is remarkable, that it coexists very frequently with tubercle in the same substratum. (*Vide* Tubercle in Serous Membranes.)

Where the hemorrhagic exudation-process borders upon actual hemorrhage it is manifest, and so is also the mode in which they are to be distinguished. The process is seen upon all serous and synovial membranes, but especially on the pleura and peritoneum; it is met with also in the pericardium, and in the tunica vaginalis testis; and, amongst synovial membranes, principally in the knee-joint.

The thickness and consistency of the hemorrhagic exudation are proportioned to the quantity and plastic properties of the fibrin it contains: it forms a peripheral coagulum, which cleaves to the walls of the serous cavity, and may contain more or less coloring matter, or may be white. The red fluid effusion is enclosed within the peripheral coagulum, and out of it further plastic ingredients are precipitated. These ingredients in process of time, are converted into a very tough leather-like layer, which undergoes very little or no organization; the fluid effusion gradually assumes a chocolate-brown, a plum-sauce, or a yeast-yellow color, and becomes fatty and glutinous; while the substance precipitated from it degenerates into a loose pulp of the same color; or it deposits its coloring matter, and becomes a clear serous fluid.

The hemorrhagic effusion is but rarely absorbed, and with much difficulty when it is. The reason of this difficulty is sometimes the continuance of a chronic inflammation, and the frequent coexistence of tubercle with it, in the stratum from which it was poured out, and sometimes the density, the impermeability, and the extremely incomplete organization, of the plastic layer that surrounds it. When it occurs on large serous membranes, it usually proves rapidly fatal by the exhaustion it produces, or by its interfering with the function of important organs; and the more rapidly in proportion to the amount of general disease that coexists with it. It is, however, sometimes borne for a long period, and under favorable general and local circumstances it may be diminished by absorption. If, in the most favorable case, it happen, that the fluid is completely removed and the peripheral laminæ are agglutinated to one another, a rust-colored, or yeast-yellow layer is found interposed between them. In the peritoneum chiefly, and more particularly on that part which belongs to the intestinal canal, the plastic layers of the hemorrhagic exudation acquire a bluish color, and after a time the color of Indian ink (melanosis stratiformis). No doubt the discoloration is owing to the action of the intestinal gas.

In course of time, the fibroid exudations frequently become the seat of calcareous deposition; and yellow, grayish, or dirty white, cheesy masses of various sizes are not unfrequently found in the cellular, as well as in the fibroid, layers of exudation. They are portions of plastic exudation which have not been organized; degenerate fibrin, which, either decaying further and becoming puriform, excites inflammation and actual suppuration and ulceration in the neighboring tissues, or else changes into a chalky concretion.

The termination of inflammation of serous membranes in *suppuration* has been already considered.

Sloughing or necrosis of serous tissue is very rarely met with as a consequence, or degeneration, of an inflammatory process, but it frequently results from the membrane being stripped of its subserous tissue, when that has been destroyed by suppuration or sloughing; or from pressure, stretching, or strangulation of the membrane; or when adjoining structures are also sloughing, or have become gangrenous some time previously, as in the instance of gangrene of the lung. Sloughy serous tissue forms a dirty yellowish, or a whitish, soft eschar, as is seen in the case of perforating ulcers of the stomach or intestines, or of strangulated herniæ; or else it is a loose shreddy, grayish, or blackish-brown, moist, infiltrated and pulpy mass, which is traversed by a dirty white thready tissue; it has the odor peculiar to slough.

The termination of inflammation in *tuberculosis*, or rather the metamorphosis of its product into tubercle, will be considered presently.

The state of the tissue beneath the serous membrane during inflammation is a point of considerable practical importance. The condition of the subserous cellular tissue, as has been already mentioned, is an integral part of the whole process. The more intense the inflammation is, and the longer it continues, so much the more do that and the adjoining tissues take part in it. Inflammatory products of various kinds are deposited in all of them, and become infiltrated, or give rise to diffused or circumscribed suppuration, or to chronic, and very considerable, thickening. The most important part is that which is taken by adjoining fibrous tissues, whether aponeuroses, capsules, or ligaments. Another remarkable fact is, that the tissues lose their vital contractility: muscles, under such circumstances, become paralyzed and lose their color. Very intense and chronic inflammation leads at last to atrophy of the subserous structures, partly by the change of texture which is produced by the inflammation, and partly in consequence of their protracted palsy. The viscera contained within an inflamed serous sac are displaced and compressed to a degree and extent corresponding to the quantity and the position of the exudation; and when this is long continued, they undergo various changes of texture, which may be included generally in the terms atrophy, obsolescence, obliteration.

Whichever of the forms that have been described the inflammation assume, it may be general in its extent, or only partial and circumscribed. It is remarkable to observe, that usually—though there are various exceptions to this rule—the parietal layer of serous sacs suffers more than the visceral, and that, therefore, the plastic exudations on it are the thicker.

3. *Softening of serous membranes.*—There is no such disease as primary softening of these membranes: when it does occur, it is consecutive, and in the peritoneum and pleuræ, ensues upon prior softening of the stomach, intestine, œsophagus, and lungs. The mode in which the serous membrane suffers is the same as has been described in the account of those diseases.

4. *Adventitious growths.*—Some of these have been mentioned as products of inflammation. There remain to be noticed:

a. Lipoma.—This occurs in subserous cellular tissue, but is an unusual disease; it consists generally of a small and lobulated mass of fat, which projects into the cavity of the serous membrane. A somewhat remarkable form is that described by J. Müller under the name of *lipoma arborescens;* it occurs on synovial membranes, especially in the knee joint.

b. Cysts.—These, on the whole, are rare growths on serous membranes; though there are exceptions to such a rule in the instance of some serous membranes, and even of particular regions and particular prolongations and duplicatures of them. Thus in the peritoneun, for instance, cysts are frequently found on that part which invests the sexual organs of the female, especially on the broad ligaments and peritoneal coverings of the ovaries and Fallopian tubes; on the great omentum the same is the case, and on the tunica vaginalis of the testicle, which in this respect bears a remarkable analogy to the sexual portion of the peritoneum in the female. Equally remarkable is the fact, that when cysts are formed on serous membranes, it is chiefly on portions connected with organs in which cancer is of frequent occurrence.

There are two different ways in which cysts are developed; and in this respect their development is analogous to that of secondary cysts upon and within anomalous serous and fibro-serous membranes. In one case, the cyst is formed upon the inner free surface of the serous membrane, and for the most part is a vesicle with very delicate walls: such cysts are sometimes very numerous; they are usually of small size, and have broad bases, rarely being attached by a pedicle. In the other case it is formed deeper in the parenchyma of the membrane or in the subserous cellular tissue—in the wall of the serous sac, and is often situated as in the broad ligaments, between two serous layers; it thrusts the membrane before it, and at length falling into the cavity, remains suspended only by a serous cord or pedicle, which is sometimes several inches long. Cysts of this kind very often have thick walls, and frequently attain a considerable size: they occur singly or in small numbers, and almost only in the neighborhood of the internal sexual organs of the female; those which have long pedicles are often observed at the fimbriated extremity of the Fallopian tubes.

The contents of the cysts are most frequently serous; sometimes they are thin and watery, sometimes thicker and mixed with albumen or with a fluid like synovia; occasionally other substances are found in them, which may be colorless or colored, gelatinous, like gum or glue (colloid), or fatty. Cysts with contents of the last-mentioned kind are often found in the omentum, and sometimes, besides the fat, they contain also hair, bones, and teeth.

c. Fibroid tissue.—One form in which this tissue presents itself, is that of *condensation* of the serous membrane, and of the cellular layer beneath it: it assumes the appearance either of milk-white, more or less circumscribed stains, which, after a time, become smooth or uneven plates of various thickness; or of bluish-white, tough, separate granulations (so called *cartilaginification* of serous membrane). Another form is that of fibroid *exudation* upon the inner surface of the membrane. The present head might include also the concretions which are found free in serous and synovial membranes, but they will be treated of in a subsequent part of the work. Moreover various fibroid growths found in the synovial membranes of joints, are formed from exudation accumulated in particular spots; they are villous and laminated, or they form clusters of small subovate bodies that resemble melon-seeds (Mayo). Bursæ are sometimes filled with balls of exudation which are undergoing a change into fibroid tissue.

d. Abnormal bony substance,—Ossification, as it is called, *of serous membranes.*—This, like the adventitious substance last described, is found as a subserous formation, on the outer side, and in the substance of the serous coat, after it has undergone a fibroid condensation of its tissue; and it also occurs in fibroid exudations upon the inner surface of the membrane. Its usual form is that of nodulated plates or cords of various size and thickness. It appeared to Meckel to be the result of an endeavor to convert a membranous into an osseous cavity, similar to that which prevails in the vertebral and cranial cavities. The granular and stalactitic form is less common; but both are sometimes found together at the same spot. Lastly, some shapeless concretions are met with, which are the chalky residue of fibrinous effusions.

The frequency with which serous membrane becomes the seat of ossification is a matter of much variety, depending on the different frequency of the changes of texture which precede it. Ossification occurs chiefly on the pleura, where it is remarkable for its extent and thickness. In the peritoneum it is almost confined to certain investing portions, especially to that covering the spleen; it occurs in the tunica vaginalis of the testicle, and, in the synovial system, sometimes in bursæ.

e. Tubercle.—Tuberculosis of serous membrane.—Tubercle affects chiefly the larger divisions of the serous system; the peritoneum, pleura, and pericardium. It is ordinarily the product of a general constitutional disease, which has been already localized in some parenchymatous organ, and in this sense the tuberculosis of serous membranes usually has some definite starting-point, or prior cause (Ausgangsherd). It sometimes, however, occurs independently of any such previous and causal deposition, and is the primary and only local affection in which the general tubercular diathesis expresses itself. It is, with very few exceptions, the result of a high degree of the general disease, and hence is associated with tuberculosis occurring simultaneously with, or soon after, it in organs which stand in immediate connection with the membrane. The starting-point for tuberculosis of serous membranes is, in general, a previous affection of the absorbent glands, or of the lungs; that for peritoneal tubercle is tuberculosis in the abdominal lymphatic system, in the internal sexual organs of the female, or in the intestines; the cause of tubercle in the pleura and pericardium is found in the bronchial glands and lungs; tuberculosis of the tunica vaginalis testis has the starting-point in the lymphatics of the genital organs, and in the testicle itself; and so on. Peritoneal tubercle is, almost as a rule, associated with the same disease in the spleen, or liver; that of the pleura with recent deposition of tubercle in the lung; and further, tuberculosis not unfrequently appears in nearly all the serous membranes at once, or almost at once, and either in one and the same form, or in the various forms to be described presently.

In some few cases the tubercle may occupy the tissue of the membrane itself, and the subserous cellular structure. Generally, however, its site is manifestly the free, smooth surface of the membrane, or it is seated quite within the surface in a false membrane of cellular or cellulo-serous structure that lines the serous membrane. In the former case it may be stripped or broken off from the serous membrane, and leaves behind it a spot of corresponding size, dull, lustreless, often distinctly opaque, and deprived of its epithelium. If it have been of large size, its pressure may have formed a pit, and then it appears as if it had been seated in the tissue of the serous membrane itself.

Tubercle presents itself upon serous membranes in the following forms:

α. One form is that of the gray, semi-transparent, crude, granular tubercle, the size of which is about that of coarse sand, or millet-seed. When chronic, this form of tuberculosis may originate at several parts of the membrane. Commencing at one or more of these starting-points at the same time, it gradually extends over large portions, or even over the whole of the surface: its advance, however, is not uniform, and hence the original depositions may still be recognized by the close grouping, and by the appearance of the granulations. In the acute form, the tubercles are usually abundant, and are sown evenly and close together over the whole expanse of the membrane, or at least over a very considerable part of it. They consist of granular tubercle of the size of millet-seed, or, as is often the case, of transparent, crystalline granulations, resembling vesicles, and so fine as to be perceptible only when the light falls favorably. Acute tuberculosis ordinarily arises out of a more or less lingering (chronic) tuberculosis of the membrane: and in that case, as well as in the rarer instances in which it commences on perfectly healthy membranes, it is usually but a partial manifestation of a general tubercular diathesis, which is exhibiting itself in several structures, either together or consecutively. This fact is one of great importance, from the absolutely unfavorable prognosis which it establishes. Dropsy of the serous cavity co-exists with the tubercle, and is directly proportioned in amount to the extent of the deposit over the membrane: general cachexia and dropsy of other cavities and organs follow in the same proportion. The œdema of the serous and adjoining cellular tissues, the infiltration of the parenchyma with serum of the blood, and the loss of its color, as well as the thin fluidity and defibrination of the blood generally, are all proportioned to the acuteness of the disease.

This kind of tubercle undergoes scarcely any metamorphosis, for the local disease which gives rise to it, and still more the general, and already far-advanced, constitutional affection, prove too speedily fatal: sometimes, however, when the course of the disease is chronic, the tubercle is found here and there obliterated (obsolete).

β. An inflammatory product, deposited upon a serous membrane under the influence of a constitutional affection,—which affection is usually already localized, and very often is even manifested in established phthisis,—may undergo the metamorphosis into tubercle. The change is induced by some inherent anomaly in the quality of the product, and is effected in various ways. Sometimes the exudation in its whole thickness degenerates into a uniform cheesy, or caseo-purulent, fissured layer, which agglutinates and connects the organs contained in the serous sac to one another, and to the parietal layer of the membrane: sometimes it is partially organized and gradually converted into a cellular or cellulo-serous tissue, while a more or less considerable portion of it becomes tubercle. The layer of exudation is then found in different stages of organization, and interwoven with isolated or confluent, grayish, fawn-colored, or dirty yellow tubercles, of the size of sand, millet-seed, or hemp-seed, and often with still larger shapeless masses of tubercle. Two species of this form of tuberculosis are in several respects remarkable:

(1.) An exudation in the form of a rugged layer, for the most part of considerable thickness, and of fibro-cartilaginous firmness, which consists of a quantity of confluent granular tubercles, and of a grayish-red, moderately vascular, lardaceo-gelatinous, or grayish, pale, slightly vascular, and lardaceo-callous, substance, in which those rugged masses of tubercle are imbedded. A comparative analysis shows that the *status* of this tubercular layer, as a vascular structure, is secondary, and that it corresponds to the lardaceous infiltration and callous condensation of the tissue of mucous membranes and parenchymatous organs around tubercle, and tubercular ulcers.

(2.) In cellular and cellulo-serous tissue recently formed on serous membranes, especially on the peritoneum, there occur yellow, cheesy or fatty, brittle masses, of round or subovate form, and of the size of peas or beans: sometimes they are shapeless, and are as large as doves' or hens' eggs.

This form of tubercle also rarely undergoes any metamorphosis; as, indeed, might be expected from the high degree which the constitutional disease (the dyscrasia), the preponderating, internal cause of the exudative process, attains; but sometimes the species just noticed—(2)—is seen, on the one hand, softening and leading to suppuration (tubercular phthisis) of the serous membrane, or on the other hand, becoming chalky.

γ. Lastly, an exudation upon serous membranes, originally free from tubercle, may, at any stage of its organization, become the *nidus* of that growth,—a form which, when it is possible, is to be distinguished from that developed in the way described in section β. That such a form exists is probable from two observations, and is not opposed by any positive facts.

(1.) In chronic inflammations of serous membrane, which recur in the exudations, one of the secondary inflammations sometimes furnishes a product upon the free surface of the older exudation; and that product becomes tuberculous in the manner described in section β; that is to say, a serous membrane is sometimes found lined with an exudation, the outer and older layer of which is free from tubercle, whilst the inner—the product of a secondary inflammation of the older layer—is tuberculous.

(2.) In the cellular false membrane lining a serous cavity, especially the peritoneum, we sometimes see tubercle, usually of considerable size

—as large as hemp-seed or peas—from the highest and central point of which loose-walled bloodvessels project, and passing to the outskirts of the tubercle, sink deeply, and so are lost, or else are seen to anastomose with other vessels of the false membrane. Indeed, in a few apt cases, the tubercle is found upon close examination to be excavated by a canal or cavity, which forms the centre of this small vascular apparatus. But, in most instances, the canal is already obliterated, the circulation is obstructed, and the vascular apparatus is beginning to waste. When the atrophy is accomplished, the tubercle is found imbedded in cellular tissue, which is streaked with blackish-blue lines. Such an appearance may give rise to the assumption that tubercle is supplied with bloodvessels, especially as I have recommended serous membranes as the structure best adapted for the study of tubercle, because in that system it may best be followed in all directions. The appearance, however, may be safely explained in the following manner: The tubercle is thrown out under the influence of a tubercular diathesis by the vascular centres which are forming in the false membrane, and arranges itself around them: the more abundant—the larger—it is, so much the more prominent does it render the vascular apparatus that radiates from its centre.

The tubercle formed upon serous membranes is frequently a hemorrhagic product; especially when it is a result of the processes just described under sections (1) and (2); indeed this is sometimes the case when it is thrown out by a primary exudative process.

The congestion that attends its production not unfrequently degenerates into inflammation, and that, for the most part, furnishes a hemorrhagic exudation, in the same manner, but not to the same extent, as the inflammation of a false membrane in which tubercle is forming. As has before been explained, the hemorrhagic nature of the exudation is owing to the fact of the blood being impoverished in fibrin by the exudation of tubercle, and also in the second case to a local circumstance, viz. the imperfect formation of the vessels in the false membrane.

f. Cancer.—Serous membranes are often perforated by malignant growths which originated externally to them: the pleura is invaded by masses of cancer deposited in the mediastina, and by large exuberant growths in the mammæ; and the peritoneum, amongst other growths, by those which Lobstein has named "Retro-peritoneal." But cancer appears on these membranes as a primary disease also. As a general rule, its appearance there has some connection with the existence of cancer in an organ adjoining, or contained within, the serous sac; so that it always shows the cancerous cachexia to be very far advanced.

The most common forms of cancer found on these membranes are the areolar and the medullary; the latter having not unfrequently the melanotic character. It consists either of laminæ, which vary in extent and are unequal in thickness; or of small nodules, like tubercle, which germinate in the tissue of the serous membrane; or of larger knots and tuberous masses, which shoot forth from the tissue over the surface of the membrane. Moreover, upon the serous layer of the dura mater, numerous morbid growths, allied to medullary cancer, are found, the internal and minute construction of which presents very much variety.

In large serous cavities, such as the peritoneum, there are somewhat rarely found very large adventitious growths, which have the same general characters as those under consideration, but are very loosely connected with the serous membrane by one or a few points, or are even entirely unattached.

If, as often happens, inflammation should attack a cancerous growth on a serous membrane, the result is a hemorrhagic exudation: the explanation of the occurrence is found in the very imperfect state of the vascular apparatus involved in the inflammatory process. (Compare what has been said as to the local causes of hemorrhagic exudation.)

g. Anomalies of secretion, and morbid contents generally.—Free *gas* is not unfrequently found collected in different quantities within serous sacs. It is met with chiefly in large serous cavities, such as the pleura and peritoneum, and its presence is due to the escape of atmospheric air from the air-passages, or of intestinal gas from the bowel. It is occasionally produced by the decomposition of ill-constituted and long-stagnated effusions; or it may be a product of the exudative process itself. In a few cases, it may even be a morbid secretion (exhalation), from the serous membrane during life.

Besides this, and the various products of inflammation already described, there occur also collections of serous fluid, and of blood.

Collections of *serum* constitute dropsy of serous and synovial sacs, and of bursæ (Ganglia); the quantity of fluid varies, and with it the enlargement of the cavity; its color, too, and its consistence and composition, especially in respect to the quantity of plastic material it may contain, vary considerably. The remarks already made upon dropsy in general apply also here.

The effusion of *blood* into serous cavities—actual hemorrhage—must be carefully distinguished from hemorrhagic exudation.

An account of various other effusions will be found in the chapters on the particular serous sacs.

Lastly, the cavities of serous and synovial membranes sometimes contain free loose bodies, which have various origins, and differ accordingly in their appearance and construction: those, more particularly, which are met with in the peritoneal cavity vary much in their kinds. They are found in the cavity of the peritoneum, within the tunica vaginalis testis, in the pleura, in the sac of the arachnoid, and in the ventricles of the brain; they are also particularly common in several of the synovial cavities, especially in the knee, and in bursæ (articular mice); they even occur in anomalous serous sacs.

Their usual size varies from that of a millet-seed to that of a pea or a bean: it is an exception to find them larger, but they do sometimes reach the bulk of a walnut: they are generally round or oval in shape, but pressed somewhat flat; sometimes their figure is irregular. They are mostly firm and elastic; and from the smoothness of their covering, which glistens like a serous membrane, they acquire a polished appearance, but sometimes there are rough and villous spots upon them.

With regard to their origin, the observations of Laennec and Béclard prove that some of them originate outside the serous membrane; while the internal construction of many others indicates that they were formed within its cavity.

The first kind includes the fibroid and fibro-cartilaginous concretion
some of which contain bony nuclei. They are originally developed i
the subserous cellular tissue, or in the serous tissue itself; but as the
gradually force the membrane before them, they become invested wit
a prolongation or duplicature of it, which remains connected with th
rest of the membrane only by a pedicle; at length the pedicle bein
worn away by friction, the cartilage falls loose into the serous cavit
It has a proper serous covering, which often bears a trace of thi
mode of development in being deficient at the spot where it was sepa
rated from the pedicle: it is then completed by loose shreds of cellula
tissue.

Those of the second kind are the fibrillated and albuminous coagulation
and precipitates from morbid effusions. They are distinguished b
their uniform smoothness throughout, by a delicate albuminous investin
membrane, and frequently by their manifest arrangement in concentri
laminæ.

Moreover, free bodies of a different nature are sometimes found pa
ticularly in the peritoneal cavity. Some of them are obsolete portion
detached from the omentum and appendices epiploacæ, which, within
bluish, gray tunic, contain fat that resembles tallow or spermaceti
others are tubercles which have become loose, and which, like the forme
may become the nucleus of albuminous coagula: whilst others again ar
fibroid, or are allied to the fibroid, tumors formed beneath the perito
neum in the uterus or its appendages, but afterwards set free.

PART VI.

ANOMALIES AND DISEASES OF MUCOUS MEMBRANES IN GENERAL.

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ANOMALIES AND DISEASES OF MUCOUS MEMBRANES IN GENERAL.

§ 1. *Defective and Excessive Development.*—Congenital deficiency of mucous membrane involves deficiency of the apparatus which it com- oses, or which, as the expression is, it invests or lines: always, there- ore, when the mucous membrane is absent, the whole apparatus is absent oo. The only instance of acquired deficiency is a partial loss of sub- tance, and it varies in character considerably according to its cause.

Preternatural development is sometimes an original anomaly, which ay be exhibited in a congenital excess in the length and capacity of ucous canals and cavities, in the existence of unusual appendages and uplicatures, or in the unusual size of prolongations and folds which aturally exist in the membrane. Sometimes such an anomaly is ac- uired: it is exemplified in the similarity that exists between the surfaces f abscesses and fistulæ, and mucous membranes; in other words, as Otto escribes it, in the development of anormal cavities and canals, which, ke normal mucous membranes, are connected, or about to be connected, ith the surface of the body: it is further illustrated by the restitution f lost mucous membranes.

In regard to the former, whether the lining membrane of the abscess r fistula be composed of cellular tissue, of serous membrane, or of any ther structure loosened in its texture so as to resemble cellular tissue, is at first a granulating vascular layer closely connected with the sub- cent structures; but afterwards it become a more distinct membrane, nd may be isolated from them: its free surface may be smooth, or may e covered with shreddy appendages and prolongations. In its structure has a general resemblance to mucous membrane; but inasmuch as it as few follicles and no actual villi, it is rather like those of simpler or- anization, such as the ducts of glands. Moreover, it exhibits patho- gical changes, which are very similar to those of normal mucous mem- ranes: sometimes it is pale, and sometimes it is found injected, red- ened, and swollen, just as is the case in acute or chronic inflammations f a natural membrane; polypus-like prolongations are formed upon it, nd fungous growths of various kinds; and further, the cellular tissue eneath it becomes thickened and callous, &c. And just as mucous sur- ces never unite together, except after some solution of their continuity, also the allied anomalous mucous canals can only be closed by laying bare the tissues beneath them, either by laceration, or by compression carried to the extent of producing atrophy.

There is a difficulty in the regeneration of mucous membranes in their original form, proportioned to the complexity of structure of the membrane that has been lost, to the amount of its substance removed, to the extent to which the submucous tissues have been likewise destroyed, and lastly, to the change of texture which those tissues have undergone during the process by which the loss of substance was occasioned. Regeneration is extremely difficult, therefore, in several respects, but it is most so after deeply-extending ulceration. We shall again have, as we have already had, occasion, when considering the mucous membranes in particular, to observe several most interesting peculiarities in the mode of repairing losses of substance, especially those occasioned by ulceration. In general, the repair of a breach of substance on a mucous membrane is effected in the following manner:—The exposed submucous structures are first condensed to a serous or fibro-cellular (callous) tissue by a reactionary inflammation of more or less activity, and then are gradually covered by the adjacent mucous membrane, which is drawn in, and attenuated as it is drawn, from the margins towards the centre of the defective spot: no actual regeneration therefore, no new growth of mucous membrane, takes place. But occasionally the provisional serous membrane that covers the defect is converted into mucous membrane; and in the intestinal canal especially, when the typhous process has been limited by the submucous tissue, that tissue becomes developed at the middle of the ulcer, even to a villous mucous membrane. Extensive and deeper losses of substance are permanently replaced by a callous (cicatrix) tissue, that only occasionally obtains a smooth covering like serous membrane. And the cicatrix is of course more dense and thicker, and the mucous membrane upon it more firmly fixed to the submucous tissues, in proportion to the amount of damage those tissues have sustained, either at first from the loss of substance, or subsequently from the reactionary process which was called forth.

§ 2. *Deviations in the size—superficial area—and in the form of mucous membranes.*—The anomalies which may be included under this general head, are the partial dilatations of mucous cavities and canals, and those which relate to the thickness of the membranes.

The former are the diverticuli spuria, as they are called, or herniæ of mucous membrane. They occur chiefly in the intestinal tract, in the urinary bladder, and also, but less frequently, in the trachea and bronchi. The mucous membrane protrudes, in the form of a rounded, pear-shaped, or cylindrical, saccular appendage, through the separated fibres of the fleshy coats: the appendage is attached by a sort of neck, and the two cavities communicate with one another by a narrow opening, which at first is a mere fissure, or is lozenge-shaped, but afterwards becomes circular, and is bounded by a kind of sphincter.

The *thickness* of mucous membranes may be increased or diminished. Permanent increase of thickness is due not only to various changes of texture, but also to hypertrophy: diminished thickness is a result of atrophy. Either may involve the entire structure of the membrane, or may affect one of its component parts only, such as the follicular apparatus, or the papillæ. Hypertrophy is for the most part produced by well-marked, and either repeated or continuous, states of irritation or of inflammation: it presents several degrees, and I shall treat of it further at a place where its development from these conditions can be more conveniently shown.

Atrophy very rarely occurs as a spontaneous affection in any mucous membrane: it must be distinguished from the softening of mucous tissue, which comes on after exudative processes. The membrane becomes more or less attenuated, and may be easily torn; its folds waste, or with the follicles and villi altogether disappear; its surface is pale and smooth, and glistens like a serous membrane. A similar attenuation is observed in the mucous membrane surrounding various extensive losses of substance, both during and after their repair. And there is yet another instance of the same condition, in which certain parts of the mucous system not only become extremely thin, but also undergo a change of texture: it is a consequence of the gradual and excessive distension which is produced by the accumulation of matters secreted during some occlusion of the cavity. This subject will be further considered.

§ 3. *Diseases of texture.*—Both acute and chronic diseases of mucous membrane are, as is well known, exceedingly frequent; and hence, as well as from the manifold connections which they maintain, both in their origin and consequences, with other systems and organs, they are diseases of great importance. For the most part, they are the result of that sensitiveness to all changes in the material components of the fluids,—whether immediate (primary), or produced through the medium of the nervous system,—as well as to all deviations from the proper evolution and distribution of the imponderable principles, which attaches to mucous membrane, as the most vascular of all structures, and the chief organ of absorption and secretion. Hence almost all acute (febrile), and many chronic, constitutional diseases establish themselves in various ways, the former rapidly, the latter gradually, upon these membranes. There are various processes, irritative and inflammatory, by which this is accomplished; but in the present chapter only the cardinal forms will be treated of, the catarrhal, the exudative, and the pustular; the other specific processes of the same class will be found described amongst the diseases of the separate portions of the mucous system.

1. *Hyperæmia, apoplexy, hemorrhage, anæmia.*—Mucous membranes are sometimes actively congested, either in consequence of some direct irritation, or from a special relation of the constitution of the blood to a particular portion of the membrane: sometimes the congestion is passive, and occurs as a consequence of marasmus and adynamia, particularly in the tracts of membrane lining the respiratory organs and intestinal canal. Again, it may be mechanical, and extend over large areas, and even over the whole of large divisions of the mucous system: the congestions which are found in the respiratory organs and intestinal canal, in diseases of the heart, lungs, and liver, are of this kind.

It presents various degrees. In the ordinary and slight degree, it either entirely disappears after death, and the membrane, whatever may

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have been its state during life, is found pale; or the ramifications of veins, or perhaps the capillary vessels, are full of blood, and the membrane is red and injected. When it has been acute, it leaves the membrane swollen and relaxed, and more or less evidently succulent, while the mucous and submucous tissues are slightly œdematous; when chronic, it occasions thickening and hypertrophy of the membrane, and a permanent increase of its secretion of mucus.

In a higher degree, the congestion advances to vascular apoplexy, and apoplexy is followed by bleeding into the parenchyma, and from the surface of the membrane: the more rapidly the congestion has arisen, or been augmented, the sooner does the hemorrhage take place. These occurrences are met with chiefly in the bronchi and alimentary tube, where they may arise either from active or passive or mechanical congestion. The mucous membrane appears red and swollen, from its injected capillaries standing thick together; or dark-red and tumid, from injection that cannot be distinguished from effusions of blood into the parenchyma; or, lastly, more or less blood is found upon its surface, or collected in the cavity which it encloses, while it is itself either in one of the states just mentioned, or collapsed, pale, and bloodless.

It must, however, be remarked, that bleeding from mucous membranes in general, excepting that from the bronchial membrane, is rarely the result of mere congestion; for the most part, and in the case of the stomach and intestinal canal especially, the hemorrhage proceeds from some other part of the membrane, which is diseased in texture, though it very often appears quite trifling in extent, or may be so small as to be scarcely discoverable.

Anæmia in mucous membranes is the result of diminution of the general mass of the blood, and especially of loss of blood by hemorrhage; it is, therefore, only a local symptom of general anæmia. The pallor which ensues, under such circumstances, especially in the mucous membrane of the intestinal canal, where it proceeds chiefly from gelatinous softenings, presents a remarkable waxen hue, and a yellowish shade of color.

2. *Inflammations.*—*a. Catarrhal Inflammation.*—This is the common inflammation of mucous membrane; it is sometimes an ordinary catarrh, resulting from the known atmospheric influences; sometimes it is the local expression of a constitutional disease, and is then a specific catarrh, either exanthematous, typhous, impetiginous, gouty, or the like: occasionally it is produced by direct mechanical or chemical irritants, &c.; moreover, it accompanies the various processes of ulceration and new growth that take place upon mucous membranes, varying in such cases both in intensity and in extent. Its course is sometimes acute, sometimes chronic.

a. Acute catarrhal inflammation.—The anatomical characters of this disease are as follows:

(1.) Redness, which varies from a pale rosy tint to a deep red: it gradually diminishes towards the margin of the inflamed spot, and then passes into the natural color of the tissue.

(2.) The injection may involve merely the finer ramifications, or it may amount to a complete distension of all the vascular apparatus, and will, therefore, vary in each membrane according to the special arrange-

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ment of its peripheral vessels. To the unassisted eye, the membrane then appears uniformly saturated with red.

(3.) Even with a slight amount of reddening and injection, the inflamed membrane loses its transparency, and becomes cloudy.

(4.) Its tissue becomes filled with an opaque, grayish, or a sanguineous grayish-red fluid, and the membrane appears swollen: the papillæ and mucous glands being in the same condition, its surface seems warty or papillary, and uneven. Sometimes the epithelium is raised in delicate, translucid, miliary vesicles, which are filled with a serous fluid.

(5.) It may be easily torn, and readily separated from the structures beneath. These structures, and especially submucous layers of cellular tissue, are loose, filled with a serous or sanguineo-serous fluid, spotted here and there with small extravasations of blood, and fragile.

(6.) At the commencement of the inflammation, the secretion exceeds the natural quantity, and is watery: as the inflammation advances it diminishes in amount, and becomes opaque and viscid: at the acme of the inflammation, it ceases altogether. After this it is gradually restored again, and is frequently streaked with a little blood: it then assumes a purulent appearance, and becomes very abundant: and remains in this condition for some time after all other marks of the inflammation, even the swelling of the tissue, have subsided. This is especially the case in mucous cavities.

Moreover, inflammations of very great intensity deposit a more or less plastic exudation upon the free surface of the membrane: the miliary vesicles upon catarrhal mucous membranes which were mentioned above, arise from this cause.

Acute inflammation often has a marked tendency to return upon slight occasions; severe attacks of it frequently terminate in superficial suppuration, which may even continue habitually. Not uncommonly it becomes chronic.

β. *Chronic catarrhal inflammation.*—The anatomical characters of this form of inflammation are,—

(1.) A dark, dull redness inclining to brown, injection, and a varicose state of the vessels.

(2.) Increase of bulk; the mucous membrane becomes thick and tumid; the swelling of the papillæ and follicles renders its surface uneven, especially if the process have been of long duration and the glands be abundant; its tissue, becoming denser and more compact than natural, is hence also—

(3.) Tough and resisting, and is with difficulty torn: it is more firmly connected, too, with the subjacent tissues, and they become swollen, dense, and tough (hypertrophied).

(4.) The secretion is a grayish or yellowish-gray, opaque, viscid mucus.

Chronic inflammation usually leaves behind it a permanent tumefaction, or hypertrophy of the mucous membrane, and a continual excessive secretion of a grayish-white and milky, or of a glassy transparent pasty mucus,—a blennorrhœa, which may or may not be attended with an exuberant formation of epithelium, and in which, accordingly, the epithelium is either rapidly thrown off from an almost bare, and, as it seems, excoriated mucous membrane, or accumulates over the whole, or over parts of the surface, and thus forms a complete laminated covering for it, or patches of various thickness here and there upon it.

The hypertrophied membrane itself is pale, or more commonly of a rusty brown, or slate-gray, and after a time, of a dark-blue color; it is thick, compact, and firm: when it is uniform in thickness its surface is smooth; sometimes, from the great increase in the size of its papillæ and follicles, it is warty and rugged; and lastly, even various duplicatures and prolongations may be formed upon it.

The two last-mentioned inequalities of the membrane are permanent, immovable folds of the membrane: they constitute what is called the mucous or cellular polypus, or the vesicular polypus.

These polypi are processes of the mucous membrane, of various thickness and length. In shape they are spheroidal or elongated, or like ninepins or cylinders; and their free extremity is thick and blunted. The mucous membrane and the tissue beneath it becoming hypertrophied at particular round circumscribed spots, form a somewhat flattened convex tumor, and progressively change into a honeycombed cellular tissue. Little by little the tumor drops into the cavity of the organ, dragging with it the surrounding mucous membrane, by which, as by a comparatively thin, and more or less elongated pedicle, it remains attached. The polypus then consists of a cylindrical prolongation of mucous membrane, which contains a cord of submucous tissue, and of a truncated extremity or knob, at which the tissue proceeds to form itself into a honeycombed cluster of vesicles and follicles, and becomes lobulated like a cauliflower; it presents a system of dilated capillary vessels; now and then it becomes turgid; it secretes a jelly-like mucus in its interstices, and when that is discharged, it shrinks.

Polypi do not occur with equal frequency on all mucous membranes. They are especially frequent upon those membranes, and parts of membranes, that are bulky and thick, and have abundance of follicles, and that are frequently attacked with catarrh. Such are the Schneiderian membrane, the mucous coat of the stomach, especially its pyloric half; that of the large intestine, particularly of the rectum; and the mucous membrane of the uterus, more especially about its cervix. The cellular polypus occurs, but less frequently, in the pharynx, the larynx, and œsophagus, in the small intestines, the urinary bladder and urethra,—though it is somewhat frequent in the female urethra; it is extremely rare, and indeed almost never occurs, in the trachea and bronchi, in the Fallopian tubes, and in ducts generally.

The catarrhal origin explains why they occur in such great numbers, whether separately or, as they mostly exist, in clusters, upon one mucous membrane.

In their thrusting the mucous membrane before them as they enlarge, and in their even protruding into a cavity, and hanging in it by a pedicle of mucous membrane, the form of the polypus is often imitated by various new growths in the submucous structures, especially by lipoma, fibroid tumors, and even by cancer. Several of these new growths have been distinguished from the mucous polypus by the names of fibrous and fleshy polypi.

A point of some importance is the condition of the submucous tissues during catarrhal inflammation and blennorrhœa. The increased sensibility of the mucous membrane gives rise to very frequent reflex movements in those which are irritable, and when the course of the process is chronic, they become hypertrophied, as well from the permanent increase in the quantity of fluids arriving in them, as from the process itself. At length, if there be much hypertrophy, the irritable and contractile submucous tissues gradually become paralyzed, and their respective cavities and canals are permanently dilated.

Moreover, chronic catarrhal inflammation sometimes terminates in suppuration and ulcer,—an event which more frequently occurs, and with more rapidity, when an acute inflammation supervenes. In that case the redness becomes more vivid, and seems as if it were identified with the mucous membrane, while the membrane itself is changed into a friable tissue, is swollen with blood, and resembles a sponge, or a spongy gland. Matter appears, either extended, as a more or less smooth coating, over the surface of the membrane, or collected in small quantities in its substance; and in this manner the tissue gradually disappears,—the whole process constituting the catarrhal (simple) suppuration, or catarrhal phthisis of mucous membrane. It leaves behind it an ulcerated breach of substance, corresponding in size to the extent of the inflammatory process,—a catarrhal ulcer, which may be limited by, or may extend deeply into, the adjoining submucous tissue. If, in the former case, the ulcer be small, it heals readily, its base becoming a dense cellular tissue, and the surrounding mucous membrane being drawn in, and at length becoming adherent, over it. An ulcer of larger extent acquires a fibro-callous base, but does not cicatrize; it remains bare, and sometimes obtains a smooth covering like serous membrane: in canals with soft walls its tendency to shrink occasions strictures; and it often, from the application of various powerful agents, becomes the seat of chronic inflammation or of gangrene, sloughs away, ulcerates anew, &c. The character of the catarrhal ulcer probably varies according to the nature of the catarrh.

Both acute and chronic catarrhal inflammations, and the various processes in which they terminate, may affect the follicles of a mucous membrane principally or alone. The walls of the follicle then redden, and the parts adjoining, as well as the follicle itself, become injected, tumid, and enlarged: its secretion diminishes in quantity or is suppressed; but sometimes it is more abundant than natural, and either pours freely forth, or being retained in the cavity of the follicle, becomes inspissated, and undergoes various other secondary changes. The result of this process sometimes is a permanent enlargement (hypertrophy) of the follicle, a dilatation of its cavity, or an habitual profuse secretion of a tenacious glassy mucus—*follicular blennorrhœa.* Sometimes the process terminates in *suppuration of the follicle*, and *follicular ulcer.* It becomes converted into an abscess, which usually bursts through, and discharges itself upon the internal free surface of the mucous membrane: a small, round, crater-like ulcer is then found situated at the top of a rounded conical tumor, and having a hard base: as the suppuration of the follicle proceeds, the ulcer becomes larger and shallower, and when the follicle is quite destroyed, is encircled by a border of loose mucous membrane; it then extends superficially, or which is rarer, deeply amongst the submucous tissues.

This process is mostly seen on membranes which have follicles in abundance, and are disposed to catarrh; on that of the air-passages, for instance, particularly in the larynx; or in the intestinal canal, especially in the large intestines, where it produces very extensive devastations.

b. Exudative processes.—Processes of exudation are frequently met with in particular portions of the system of mucous membranes; but their nature is very various, as their products, and the condition of the mucous membrane in connection with them, manifest.

The best known exudative processes upon mucous membranes are those named croupy inflammations, especially those that occur in the pharynx and air-passages. They are characterized by their plastic product, which varies in consistence from that of cream to the toughness of leather, and is grayish-white, or yellowish and fibrinous; it sometimes covers the membrane at a few insulated spots, and sometimes forms a more extensive film over it like hoar-frost; occasionally it invests the membrane like a layer of gauze, while in some cases it constitutes a membranous, and very often a tubular, lining for the mucous surface.

From all analogy it is probable that, at the commencement of the process, a serous fluid is effused by which the epithelium of the diseased mucous membrane is destroyed, and that the exudation of the plastic matter takes place afterwards. This matter, the general characters of which have just been depicted, forms in a severe case a membranous coagulum, the thickness of which may vary, but not unfrequently equals, or even, exceeds, a line: towards its margin it is thinner and less tough, and it is at length lost in a layer of muco-purulent substance. At first it adheres to the mucous membrane, and on that surface which adheres to the membrane some incipient vascularity is sometimes seen in the form of small bloody points; some of these points are single, others divide into fine twigs towards their peripheral extremity. At a later period, a viscid, muco-purulent fluid is effused beneath the plastic exudation, so that it becomes loose, and is at length set free.

The mucous membrane underneath the exudation is variously tinted, but for the most part is of a very pale rosy color: it looks sore and excoriated, and is more or less swollen, and its papillæ especially are distinctly swollen; its surface is covered with numerous red, soft, bleeding spots like granulations, which correspond to the vascular points on the adherent surface of the exudation. The submucous tissues, especially the cellular tissue, appear infiltrated.

Neither during nor after the croupy process does the mucous membrane suffer any material injury to its texture; the speedy production of mucus and epithelium prevents any further organization of the plastic exudation beyond the rudimentary condition just described, and it never enters into an organic connection with the mucous membrane.

The croupy process occurs on all mucous membranes, and sometimes extends over a very wide tract. The mucous membrane of the respiratory organs shows an especial tendency to it, and we meet accordingly with laryngeal croup, tracheal croup, bronchial croup, and croupy pneumonia. In those parts, and on the inner surface of the uterus after childbirth, it is very often a primary process; while on most other mucous membranes it is only secondary, and occurs principally as a consequence and an expression of the degeneration of an exanthematous, or typhous, or some other process attended with exudation, such, for instance, as the cholera process; it arises also from pyæmia, &c.

Other exudative processes give rise, either from the first and exclusively, or else after furnishing, or whilst furnishing, a plastic product, to a loose, pulpy, puriform or sanious exudation, of a variously shaded brown and green color, and a very offensive smell. The mucous membrane, under such an exudation, softens to a pulpy, or a shreddy and crumbling mass, which has an offensive smell and the same color, or may be also dark brown, chocolate-colored, or like coffee-grounds from hemorrhage. These processes are named *putrefactive*, and may be primary, but they are much more frequently secondary.

A special form in which these exudations appear, is that of the benign and malignant aphthæ,—exudations, that is, which, at first, at least, are confined to rounded or oval spots. They are most common by far on the mucous membrane of the mouth and pharynx; they do, however, occur on all other membranes, but are then generally secondary. The process of softening that goes on beneath the exudation, occasions a loss of substance in the mucous membrane, that may be called an aphthous ulcer.

Other exudative processes, which for the most part extend over large portions, or the whole tract, of a mucous membrane, furnish products that are either albuminous, jelly-like, and pellucid; or milky, mixed with delicate fibrinous flakes, and pasty; or thin fluid, mostly serous, and of a very pale grayish-white, yellowish, or reddish color, or quite colorless. They run their course, sometimes with moderate redness and injection, sometimes with remarkable paleness of the mucous membrane, with tumefaction, infiltration, and at length softening and removal of the epithelium, with softening of the tissue of the membrane itself, and degeneration of it to a pale-grayish, yellowish, rosy, or dark-red stratum that is apt to bleed, and may be wiped off like pap, and with similar softening of the follicles. Such processes are, for the most part, secondary, and their chief seat is the mucous membrane of the intestinal tract. The most remarkable of them for extent, for quantity of product, and for the rapidity of its course, has been learnt in modern times,—the Asiatic cholera.

In very severe cases of the exudative process, the submucous muscular tissues become paralyzed: they are blanched, relaxed, and infiltrated.

c. The Exanthematous processes upon mucous membranes are allied to the exudative. They are sometimes the manifestation of a very great degree of constitutional disease, and form a complementary addition to eruptions on the general integuments; sometimes they are vicarious with the crisis of an exanthema upon the skin, which, from various influences that we are ignorant of, is insufficiently developed; and sometimes they constitute a specific eruption, arising from a special relation between the general disease and a particular tract of mucous membrane,—*mucous exanthema*, as it has been lately denominated. The seat of the two former kinds is chiefly mucous membrane where it adjoins skin, but to a certain extent also it is found where mucous membrane is connected with the original "*atriis morbi;*" such as the lining of the mouth, pharynx, tracheal passages, conjunctiva, or urethra: the last kind, on the contrary, is confined to particular parts of the mucous system, as to the ileum in typhus, and the colon in dysentery.

The following are the forms observed: diffused or circumscribed redness or spots,—*erythema* which sometimes, by their various tints, betray the kind of constitutional affection that exists; *vesicles* of various sizes and number, separate or confluent, and filled with a fluid that is chiefly serous, but passes through sundry changes as the process goes on: and *pimples* and *pustules* of different dimensions. As belonging to this class, we may enumerate erysipelatous affections of the pharynx, especially those which take place in scarlatina, and in its anomalies; the miliary eruptions that occur upon mucous membranes affected with catarrhal inflammation, or at the commencement of dysentery; the affections of the mucous membrane of the larynx in measles; the pustules of variola on the pharynx and air-passages, and on the urethra; and herpetic pustules. In many processes, generally enumerated in this class, which assume the form of papules and nodules on the membrane, the principal seat of the affection is the follicular apparatus, as is the case in true intestinal typhus, and in several other processes allied to it.

Exanthemata upon mucous membranes pass, in favorable cases, to the same terminations as the corresponding processes upon the integuments; but in acute cases, in which, from any cause, the exanthematous process is concentrated upon one section of the mucous system, it may readily occasion softening of the membrane, and loss of substance by ulceration of its tissue. Of this kind are the ulcerations that occur, rarely indeed, on the mucous membrane of the throat and larynx during and after scarlet fever, measles, and variola; as well as the softening of the mucous membrane of the large intestine that constantly accompanies severe cases of dysentery, and the peculiar metamorphosis that almost invariably takes place in the typhous follicle of the intestinal mucous membrane.

The study of this portion of the pathological anatomy of mucous membrane is attended with great difficulties: for, with the exception of some of the processes that have been mentioned, such as typhus and dysentery, they occur so seldom, the products of the exanthema are so delicate, and there is such loss of color and collapse of the membrane after death, that very little is known about it.

8. *Ulcerative processes.*—Ulcerative processes are very frequent upon mucous membranes; and withal very various in their forms, in regard both to the anomaly of texture which gives rise to them, the mode in which the ulcerative solution takes place, and the form which they derive from the stratum in which they occur, and from the fact of its having been the first diseased part or not, &c. However, therefore, I may think to have increased our knowledge of several of the processes connected with these ulcerations, and to have established the diagnosis of several forms of ulcer, especially upon the mucous membrane of the alimentary tube, yet not more than a few foundation-stones have been laid for a comprehensive knowledge of the ulcers of mucous membranes.

They are sometimes the result of the softening of mucous membrane which is induced by the processes just described, the catarrhal, the exudative, and the exanthematous. Sometimes they commence upon the surface, sometimes deep in the parenchyma of the membrane: they may attack the whole of a certain circumscribed space at once, or advance from a definite starting-point, as in the case, for instance, when those processes are situated in the follicles. Moreover, though sometimes the immediate result of the process, they are at other times a secondary consequence of it, being brought about by the action of matter exuded upon the free surface, as well as in the tissue, of the mucous membrane, after that matter has undergone some solution,—some change of its nature,—some metamorphosis; as is the case, for instance, with aphthæ, and with the matter of typhus.

Or again, they may be produced by the metamorphosis of some new growth which has been infiltrated into the tissue of the membrane, or of some tumor which has encroached upon it, and by the reaction consequent on that metamorphosis: of this kind are the tubercular, and the cancerous ulcer, &c.

The various ulcerative processes upon mucous membranes sometimes run an acute course, sometimes a chronic. They sometimes extend readily from the mucous to the submucous tissues; sometimes their tendency is rather to spread superficially, that is, to abrade the mucous membrane, and merely expose the tissues beneath it; sometimes like the fundamental processes by which they are occasioned, as for instance, the typhous process, their progress is limited by the adjoining submucous tissues.

This subject will be found to be pursued further where the ulcerative processes of the parts of the mucous system are separately adverted to.

4. *Œdema of mucous membranes.*—All the processes already described, especially the exudative and exanthematous, are attended with œdema; and so also is the ulcerative, in various degrees and to various distances from the actual seat of disease; but œdema may originate also in the submucous cellular tissue, in consequence of many primary and secondary congestions and inflammations which occur in it and in its neighborhood.

The usual seat of the infiltration is the submucous tissue. It generally forms a pale yellowish or grayish, translucent and tense, or a flabby and movable swelling, not distinctly circumscribed, over which the upper layer of the mucous membrane is stretched: when it is considerable, it extends through the whole thickness of the mucous membrane also, which then loses the character of its structure, and may be torn with the slightest effort.

The œdema reaches its higher degrees chiefly on membranes and duplicatures of membrane which are extended loosely and movably over thick strata of cellular tissue, especially upon the mucous membrane of the intestines and their valves and folds, and the duplicatures at the orifice of the larynx.

Its importance depends upon the processes which occasion it: it is only at certain parts that it becomes at all serious, as at the glottis, where it contracts and at last closes the orifice.

5. *Deposition—Metastasis—on mucous membrane.*—This, on the whole,

is an uncommon appearance. It sometimes presents the character of a small collection like a furuncle, and sometimes forms a flat scale over the superficial layer of the mucous membrane. It ends in ulceration or in slough, according to the nature of the poison in the blood by which it is occasioned.

6. *Mortification of mucous membrane.*—Mortification presents itself in various forms: the mucous membrane may become a grayish-white, or whitish-yellow, dry and rotten, or moist and lacerable, eschar: such is the change that results from pressure in strangulated hernia, from excessive distension and extension, or when it is separated from the subjacent tissues, through which it is supplied with bloodvessels.

Or, after having suffered absolute stagnation of the current of blood, it may degenerate into a dark-brown, or dark-greenish, shreddy friable substance, which gives out the extremely offensive smell of sphacelus, and is more or less infiltrated.

Or else, during the softening of an inflammatory product, which is infiltered through the tissue of the membrane, and generally also is combined with an aphthous exudation upon its surface, diffused, or more commonly circumscribed portions of the membrane degenerate into a shreddy and crumbling, or a uniformly pulpy mass, that is variously discolored, and has a very offensive odor.

When affected with the actual sloughing described under the first-named forms, the mucous membrane generally shares that condition with other adjoining structures.

7. *Softening of mucous membranes.*—If we exclude from consideration the relaxations of tissue that mucous membranes suffer from inflammation and œdema, and the solutions which take place during and after exudative processes, we shall still find conditions occurring on some mucous membranes, particularly in the stomach, œsophagus, and intestinal canal, which differ entirely from the former, both in their causes and in their anatomical characters,—conditions which are included within the term *softening* in its restricted sense: they will be found treated of amongst the diseases of the apparatus in which they occur.

8. *Change of texture which mucous membrane undergoes when preternaturally exposed to atmospheric air, and when long subjected to distension.*—*a.* The mucous membrane of prolapsed and everted organs is liable to the former kind of change. At first an acute inflammation attacks it and occasionally rises to considerable severity, but afterwards it becomes chronic, and at length terminates in induration. Such membranes become dark red and swollen; their secretion soon increases in quantity, and then they produce a puriform moisture: they may even clothe themselves with a plastic exudation, while underneath they appear raw and excoriated. At length the inflammation moderates, the secretion just mentioned ceases, and the redness diminishes, but the membrane remains thickened, and its texture more compact than natural; it is covered with a thick closely-adherent layer of epithelium; and hence appears dry on its surface, smooth, and glistening; its internal texture resembles that of tendon; and it acquires something of the appearance of the corium, or of a regenerated, or cicatrix, tissue.

b. The second change of texture affects the inner coat of the excretory ducts and reservoirs of glands, and of other hollow organs which are lined with mucous membrane: and the condition under which it occurs is that of some contraction or closure of the orifice, in consequence of which the secretion of the gland, or of the mucous membrane itself, accumulates, and gradually distends the cavity beyond its normal dimensions. It is observed in the gall-bladder, in the Fallopian tubes, and even in the uterus, in the excretory apparatus of the kidneys, and in the appendix vermiformis of the cæcum. This change of texture consists in a slow atrophy of the mucous membrane, and gradual condensation of the submucous cellular tissue to a serous layer, which at last takes the place of the mucous membrane. The tissue being changed, of course the secretion also is gradually altered; and instead of mucus a fluid like synovia, and afterwards a thin serum, are secreted. This condition bears generally the name of dropsy of the respective organs,—dropsy of the gall-bladder, of the Fallopian tubes, of the uterus, &c.,—*dropsy of the excretory ducts of glands.* The membrane which usurps the place of the mucous structure is thenceforward subject to the diseases of serous membranes in general; and some of them are remarkable as not occurring to normal mucous membrane, or to submucous cellular tissue; such for instance as ossification.

9. *Adventitious formations.*—Strictly speaking, very few adventitious growths are developed in and from the parenchyma of mucous membranes themselves: for, with the exception of teleangiectasis, tubercle, and cancer, and of these indeed, only particular conditions and forms, almost all the new growths belong to the submucous cellular tissue. But as that tissue is intimately connected with the mucous membrane over it, so are also the new growths that originate and spread in it. The mucous membrane becomes involved in various ways, as may be deduced from the following remarks. There are, moreover, several other affections of the same class to which mucous membranes are subject only after having undergone a previous change of texture.

a. Growths of horn and hair have, in a few cases, been seen upon different mucous membranes, particularly on the conjunctiva, the mucous membrane of the intestinal canal, and that of the urinary bladder.

b. Lipoma.—This growth is almost confined to the submucous cellular tissue of membranes near which a considerable quantity of fat is occasionally deposited. It is by no means rare in the submucous cellular tissue of the intestinal canal, especially of the small intestines; and it is met with also, but less frequently, in the stomach. It forms a rounded tumor, with a broad, or a somewhat constricted base; its size is mostly inconsiderable; it protrudes into the cavity, and is covered with the lining membrane of the organ in which it is developed.

c. Cysts are formed in cellular or other submucous tissue, but they very rarely occur. They displace and stretch the mucous membrane, and, when of large size, even produce attenuation and atrophy of it.

d. Fibroid tissue occurs as—

a. An adventitious fibroid growth of various size in submucous tissues: as such it presents itself under two forms, the second of which, for several reasons, is of much importance.

One of these forms is that of spherical, oval, or subovate, bluish-white, tough and elastic, concretions, the texture of which is very compact. It occurs in extensive tracts of submucous cellular tissue, particularly in the stomach and intestinal canal, and forms movable tumors which protrude inwards: they are very seldom larger than a pea.

The other form is that which has been named fibrous, to distinguish it from the mucous or cellular polypus; an adventitious growth of fibrous, and for the most part lax, texture, vascular, succulent, apt to swell, and generally more or less lobulated towards its periphery. It takes root by a single or by several stems, in submucous tissues of fibrous or muscular texture; it then grows towards the cavity of the organ, and thrusts before it a covering of mucous membrane. If it reach a large size, it expands the cavity on all sides; but if there be any hindrance to its increasing in the direction of the cavity it will grow principally in one direction, in either case destroying the walls of the cavity, even though they be of bone. Of this kind are the large fibroid growths, also named sarcomatous polypi, that spring from the submucous periosteum of the nares and adjoining cavities, from that of the basilar process of the occipital bone (the upper wall of the pharynx), from the perichondrium of the cartilages of the larynx, and from the innermost (submucous) layer of the substance of the uterus.

β. Fibroid tissue occurs also as fibroid and cartilaginiform thickening of the walls of mucous cavities which have been converted in the way already described into serous cavities: the fibroid tissue may then, as in the case of membranes originally serous, be deposited as an exudation upon the surface of the new membrane, or as a subserous production, beneath it. Under the same conditions, that is to say, only after the mucous membrane has undergone this complete change of texture,

e. Anomalous bone is formed upon it, or *ossification*, as it is called, takes place: and this again may be a subserous production or an ossified exudation on the surface. Cavities of mucous membrane are in this manner sometimes converted into bony capsules; but the only instance in which I have observed it is the gall-bladder.

f. New growths of cellular tissue, or *condylomata*, occur upon some mucous membranes, especially upon the female organs of generation, in the mouth, &c.

g. Teleangiectasis.—Congenital vascular nævi are, on the whole, a rare occurrence in mucous membrane, especially if those be excepted which extend from the skin to adjoining mucous textures, as, for instance, from the skin of the face to the mucous membrane of the lip. When they do occur, it is usually in the form of bluish-red, flattened or irregular, elevations of various sizes, and rarely in that of actual tumors or excrescences: they may be most frequently observed on the inner membrane of the intestinal canal.

h. Tubercle.—Tuberculosis is one of the most frequent, and, at the same time, most destructive diseases of mucous membranes: its frequency, however, is not the same in all of them. The devastations which it produces, too, though they vary considerably in their degree, are greater, on the whole, than those which result from any other process upon mucous membranes. The tubercle is deposited in the parenchyma, i. e. in the corium of the membrane, and in the immediately adjoining stratum of submucous cellular tissue,

a. Either gradually and at intervals, for the most part without any manifest congestion and stagnation, in the form of isolated or clustered gray crude granulation;

β. Or, with evident symptoms of inflammation, as an inflammatory product, infiltered into the parenchyma of the membrane, and partly also exuded upon its free surface. The product in this case, either has from the first the character of yellow tubercle about to soften; or it becomes rapidly discolored, and soon acquires that character. Large tracts of mucous membrane degenerate into a lardaceo-caseous and firm, but friable layer, and the submucous tissues become dense, callous, and thickened. Acute tuberculosis, in the form of extremely fine, transparent, and crystalline, or of opaque, wheylike, grayish granulation, seems not to occur on mucous membranes, at least, not in the marked degree in which it prevails on serous membranes, and in certain parenchymatous organs.

Precisely the same forms of tuberculosis seem to occur in the follicles and glandular apparatus peculiar to certain mucous membranes, such as those of the mucous membrane of the bowel.

Of the several large sections of the mucous system, the most frequently diseased is the intestinal tract; next come the air-passages, and after them the lining membrane of the sexual organs of the female, the seminal ducts of the male, and the urinary passages in both sexes. And it is chiefly certain parts of these membranes that are subject to the disease, as will appear in the consideration of the diseases of the several organs; for there are some parts which rarely, and others that never become tuberculous. Those to which the former observation applies, are certain portions of several mucous membranes, which, in the abundance of their glandular apparatus, approximate to the character of so-called parenchymatous organs; but still there are remarkable exceptions to this rule. Thus, in the intestinal mucous membrane, the disease occurs chiefly in the ileum, which has an extensive follicular apparatus, and in the follicles themselves; and, in the mucous membrane of the air-passages, principally at the posterior wall of the larynx, which is so rich in glands; in the sexual organs of the female, on the other hand, the cervix and vaginal portion of the uterus, and the vagina itself, all which are richly supplied with follicles, are exempt from tuberculous disease; the glandular mucous membrane of the stomach is rarely the seat of it, &c.

Tuberculosis of mucous membranes is sometimes a primary disease, as is the case particularly when it occurs on the inner coat of the Fallopian tubes and uterus; but far more frequently it is a secondary and dependent affection, occasioned by previous, and for the most part advanced, tuberculosis of some parenchyma which stands in close relation with the diseased membrane, or of some generally important parenchymatous organ, such, for instance, and, above all, as the lungs.

This condition of tuberculoses in mucous membrane, viz. their originating from a considerable amount of constitutional disease, which is already manifested by an advanced tuberculous affection of a parenchymatous organ, is the reason why tubercle in that membrane undergoes scarcely any other metamorphosis than that of softening, and gives rise to tubercular phthisis of the membrane.

The *gray granular tubercle* softens in the substance of the mucous membrane, and forms a small vomica. Opening on the free surface of the membrane, the vomica becomes a small circular ulcer, the margin of which is sometimes flabby, but usually is hard and prominent: its base is composed of a stratum of mucous membrane, or of submucous cellular tissue; and it also may be soft and lax, but it is usually callous and condensed.

This *primary minute tubercular ulcer* enlarges superficially as well as in depth, by coalescing with neighboring ulcers, and by the softening of tubercles which have been deposited secondarily, during its progress, at its border and base. It thus exchanges its original form for a secondary and still more characteristic one; for it acquires sinuous, serrated, jagged, and gelatino-lardaceous borders, and a dense callous base, beset with islands and far-jutting promontories of mucous membrane; while the tissue of the base, as well as of the borders, appears interwoven with tubercles, for the most part yellow and softening.

The *tuberculous infiltration* of mucous membrane burrows in different directions, and becoming caseo-purulent, degenerates, together with the tissue which it involves, and which has lost its characteristic structure, to a tubercular sanious matter.

From the mucous membrane, and especially from the base of the tubercular ulcer, the deposition of tubercle advances into the different submucous tissues, and gives rise to a destructive ulceration that in membranous canals and cavities leads to perforation.

The tubercular ulcer of mucous membrane very rarely heals, as may be supposed from what has been said. When it does so, it always leaves at its borders, and still more in the structures that formed its base, a permanent callous condensation, corresponding to the original size of the ulcer. But for an account of this, and of several essential peculiarities which tubercle and the tubercular ulcer present on different mucous membranes, I must refer to the description of the diseases of particular mucous organs.

i. Cancers.—Mucous membranes are very subject to cancerous affections; some are more frequently diseased than others, and especially the mucous lining of the whole alimentary tube. It would, however, be erroneous to regard every such affection as a primary affection of the mucous membrane; for in the majority of cases, the cancer originates in the subjacent cellular tissue and the mucous membrane is diseased secondarily and by contiguity.

Although any of the various cancerous growths may occur in mucous and submucous tissues, yet, so far as I am aware, nothing definite, nothing supported by numerous observations, can be brought forward to prove the occurrence of cancer primarily in mucous membrane, or to show the condition of that membrane, when the cancerous growth appears originally in the submucous tissue, except in the cases of areolar, medullary, and fibrous cancers.

The areolar and the medullary are the forms of cancer in which primary cancerous degeneration occurs in the tissue of the mucous membrane. They are rather frequent.

The areolar is known by its characteristic degeneration: it extends for the most part, over large tracts of mucous membrane, especially in the stomach and intestinal canal.

The medullary presents itself,—

a. Sometimes as nodules, which are of a round, or slightly convex form, or which even produce a navel-like depression on the free surface of the mucous membrane; they are situated in its parenchyma, but project more or less above its free surface: and they have a lardaceous, or medullary (encephaloid) appearance. Cancer of the mucous membrane in this form is scarcely ever the primary cancer in the system, but is almost always a consecutive affection, combined with some previous cancerous disease of the adjoining submucous tissues.

β. Another form of cancer which occurs more frequently, and particularly on certain mucous membranes, is looked upon most properly as a kind of medullary cancer. When in an advanced state, it forms more or less extensive spherical tumors (fungi), which are attached by a constricted neck-like base, or even what might, with reference to their bulk, be named a pedicle. They take root in the parenchyma of the mucous membrane and the immediately adjoining cellular tissue. They are for the most part loose, very vascular, abundantly supplied with blood, and of a bluish-red color; and they readily swell, and bleed frequently and severely. They are composed of a delicate membranous texture, that sometimes breaks down into fibres, sometimes into laminæ, and is filled with a whitish or whitish-red marrow, or a similar encephaloid juice. In many cases, the mucous membrane is affected in this manner at some one circumscribed spot; in other cases the growths spring up on a membrane in the form of smaller excrescences which are attached by a pedicle, and at their free extremities are shreddy and grow like a cauliflower, and are clustered so closely together, that the whole tract of membrane which they occupy seems to be degenerated into them. Their elementary texture, and their development from their mother soil, has been already described, and a special form has been mentioned as an epithelial fungus. I have also shown their alliance with the cauliflower excrescences that occur on anomalous serous membranes, i. e. on the inner surface of cystoid growths; and, lastly, have mentioned, that they no doubt constitute for the most part the erectile cancerous tumors, as they have been called, particularly by French observers.

They are often found in the mucous membrane of the stomach and intestinal canal; but they are particularly frequent within the urinary bladder. Frequently, and indeed generally, they constitute the primary cancer, that is to say, that which first appears in the organism; and they continue the only one, until by their sanious discharge and hemorrhage, they prove fatal.

Mucous membrane is always affected by fibrous cancer secondarily, being destroyed by the advance of that growth from the submucous tissues. The mucous membrane may be in various conditions: of these some have been described already, and others will be mentioned in the account of the particular organs in which they occur; but there is one character which may be spoken of in this place, which it shares with other structures that become involved by contiguity in cancerous diseases, and more particularly with the skin, namely, that when encroached upon by a mass of cancer, mucous membrane becomes adherent to it, united with it, and at length entirely lost in it.

PART VII.

ANOMALIES AND DISEASES OF THE SKIN.

PART VII.

ANOMALIES AND DISEASES OF THE SKIN.

§ 1. *Defect and Excess in Development.*—Congenital deficiency of the integuments is extremely rare, whether extending over the whole, or only over parts of the body. An instance of the former kind was observed by Bartholin; and Cordon met with a case in which the skin was wanting from the knees to the toes.

Upon the skin of new-born children there are often seen circumscribed spots, the complete development of which has been arrested by some pressure during fœtal life. It is uncommonly thin and transparent, and its defect appears proportioned to the closeness of its union with the fibrous and serous membranes beneath it. Instances of this deficiency are seen in hemicephalus, in spina bifida, and in several of the fissions of the anterior wall of the trunk. A congenital defect of another kind has also been met with, in which the general sac of the integuments is so small at particular spots as, according to an observation of Otto's, on the lower extremities of new-born children, to form strictures.

An acquired partial deficiency of skin is produced by wounds, burns, suppuration, sloughing, and other causes from which losses of substance ensue.

A preternatural growth of skin may occur as a congenital condition and produce an increase in the capacity of the general sac. The additional skin is loose and movable, and hangs in folds and appendages; thus at the end of the spine it forms a sort of tail. Instances of an acquired excessive growth of skin are furnished by several of the encysted tumors: they usually present here and there spots that resemble cutis; and they are the tumors in which the hair that grows in cysts is chiefly found.

The same class includes all cases of regeneration of skin, whether destroyed by wounds, burns, or cauterization, by the various ulcerative processes, or by mortification. The loss is in general easily repaired, but always in a form that differs more or less from the original skin. The new structure consists of a dense cellular layer of various thickness and of epidermis; but it has neither papillæ, sebaceous glands, hair follicles, nor sudoriparous glands. It is usually tightly stretched and whiter than natural: sometimes it is smooth and shining, sometimes it has a rugged and uneven, stellate, knitted, or areolar surface: very often it is but slightly movable over the subjacent structures, and occasionally is intimately united with them. It lies beneath the level of the surface of the rest of the skin.

§ 2. *Anomalies in the Size (capacity), the Thickness, and the Form of the Sac of general Integuments.*—In regard to size, the congenital anomalies already mentioned belong also to this section. Further instances of acquired anomaly in this respect are found, on the one hand, in the contractions, shortenings, &c., which result from various losses of substance and cicatrization consequent upon them; and on the other hand, in the dilatations to various amounts, which are produced, for the most part, by gradual distension or traction. Moreover loose, or soft and elastic, pendulous growths of various sizes are formed upon the skin; within an attenuated corium they enclose a delicate cellular tissue, most of which is newly formed (molluscum simplex), and occasionally they contain also some adipose tissue, which has protruded through the meshes of the deeper layer of the corium.

The form of the cutaneous sac is disfigured not in these cases only, but also in a more or less striking manner when there exist, or have existed, many diseases of its texture.

The skin deviates from its natural thickness in both directions.

An abnormal *thickness* is sometimes occasioned by congestion or inflammation of the skin, and attended with expansion and moistness of its texture: sometimes it is the result of a continuance or repetition of the same processes, in which case the deposition of their plastic product in the tissue of the skin adds condensation of texture and firmness to the increase of its thickness: sometimes the cause is hypertrophy, which again may chiefly affect the papillæ or the deeper layer of corium: and, lastly, it is sometimes the effect of the development of adventitious growths in the corium.

Hypertrophy of the skin is in many nævi a congenital affection; but more frequently it is extended over wide tracts of skin, and is a result either of stagnation in the venous or lymphatic system, or of habitual inflammatory processes, particularly those of exanthematous and impetiginous nature. It accompanies, on the one hand, under the name of elephantiasis (Pachydermia of Fuchs), exuberant growths—hypertrophies—of the subcutaneous cellular tissue; and on the other hand, most probably all, but particularly the more important, anomalies of the secretion of epidermis.

The form it presents is very various. Sometimes the hypertrophied portion of skin is smooth, sometimes the irregularity of the hypertrophy renders the surface rugged and tuberous: the skin may be movable over the subjacent structures, but in advanced degrees of the disease it is stiff and adherent, especially to fibrous structures; and the diseased part, the leg for instance, then becomes immovable; its muscles, and even its bones shrink, and the articular extremities of the latter become anchylosed.

The papillæ become hypertrophied in various degrees and forms: sometimes they resemble the villi of the intestines: sometimes they constitute excrescences which are attached by a pedicle, and truncated, or split like a tassel at their free extremity, or are sessile, rounded, and like a mushroom, &c. Hypertrophy in these forms are seen on nævi, and on portions of skin which have long been withdrawn from the atmospheric influence, and exposed to that of warmth and moisture, or have been covered with emollient and slightly irritating plasters, &c. It is noticed also in parts at which the skin has been in contact for a lengthened period with its own secretion, as it is in the deep fissures between the rolls and knots of skin in cases of elephantiasis; in the neighborhood of chronic ulcers; and on spots of skin covered with scaly eruptions: the hypertrophy of the papillæ is very marked also in decided cases of ichthyosis. Lastly, it is found,—at least it is assumed as probable,—that the genuine common wart (Verruca vulgaris) is a hypertrophy of the papilla beneath a very thick layer of epidermis, which dips in sheath-like processes into the deeper parts of the growth. Warts are exceedingly common upon the hand, especially on the fingers; occasionally too they occur in other parts, as, for instance, on the forearm.

The skin may yield to distension or traction from within, and become unnaturally *thin*. Instances of this are met with in cases of dropsy of very dilatable serous sacs, of the peritoneum, for instance, or the tunica vaginalis testis: in anasarca; or when large tumors are growing in the subcutaneous cellular tissue. The fasciculi of fibres that compose its deeper layers separate from one another, its exterior dense stratum becomes so thin as to be transparent, and even at last to suffer gradually a solution of continuity. In the fœtus, under these circumstances, it assumes the character of a serous or a fibro-serous membrane, and uniting intimately with the subjacent membranous structures, appears, as was before said, to be deficient at such spots.

When long-continued pressure is exerted upon one fixed spot of skin, such, for instance, as is produced by a tumor, complete atrophy sometimes takes place: the skin is gradually reduced to a thin vascular stratum, which secretes a viscid epidermal mucus, and at length is completely perforated.

Primary atrophy of the general integuments, strictly speaking, does not occur; but they become atrophied rather frequently as a secondary consequence of repeated attacks of inflammation, especially those of impetiginous character. The skin becomes thin and vascular, acquires a dirty brownish or bluish color, and generally gives way upon very slight injury; at last it changes into a dense white cicatrix tissue.

§ 3. *Anomalies in Consistence.*—Laxity of the corium exists congenitally in cases of nævus; and congestion or inflammation will, after birth, bring on a state in which the texture of the skin is loosened or expanded. It becomes loose also in parts which are withdrawn from the air, and continually exposed to moisture; in parts where perspiration is constantly taking place, and in the hands of little children, who have a habit of sucking them, &c. But, again, cutaneous tissue is sometimes increased in density and hardened; it becomes hypertrophied and thickened, or without being thickened, it may be hypertrophied, dry, and harsh.

A certain amount of softness of the skin, as well as of hardness and dryness, is sometimes merely an individual peculiarity of the whole organ.

§ 4. *Solution of Continuity.*—The general integuments are, in the first place, liable to very numerous and very varied mechanical injuries: solutions of continuity may also be produced in different ways by the action of chemical agents upon the skin; and, lastly, the same result ensues from the many ulcerative processes that take place in this structure. We must, however, notice the various forms of separation of the epidermis from the corium (spontaneous excoriations), that are occasioned and kept up by diseases of the skin, the spontaneous lacerations already alluded to, that are produced by extreme distension, and the fissures extending into the corium, which, in many chronic diseases, proceed from the splitting of an extremely dry epidermis (chaps—Rhagades). They heal in the usual manner, either by immediate cohesion of the lips of the wound, or by granulation and cicatrization.

§ 5. *Anomalies in Color.*—The deviations from the natural color of the integuments are very numerous. They are, in general, either an absence of color or pallor; or a deepening of it; or with one or other of these may be combined a discoloration. Sometimes they are universal, at other times they are confined to larger or smaller tracts, or even to points, of the skin, in which case they are often almost peculiar to particular regions of the body. Their site may be the cutis vera, and their principal cause an anomaly in the quantity of blood circulating in its vessels, or rather a transient or permanent alteration in the constitution of the blood; or they may be situated in the epidermis, especially in the innermost—the Malpighian—layer of it, and may proceed either from the removal of the fibrin from the elementary cells of which the layer is composed, or from their containing an excessof fibrin, or some unusual pigment. Their cause is, in some cases, an anomaly in the constitution of the blood; in others it is some external influence affecting the skin during life: but in neither case is the mode in which the cause operates fully understood.

Lastly, all diseases of the texture of the skin are, of course, attended and followed by changes of its color.

Pallor, or change of the color to a variously tinted *white*, is observed during the lack of blood that succeeds hemorrhage and exhausting diseases; it occurs in dropsy, and in a very marked degree in cases of chlorosis. In Albinoes (Leucæthiopia) it is the result of a congenital deficiency of pigment, while in Achroma, the same defect is acquired. The latter condition may be seen in Negroes, and indeed in Europeans, wherever the surface is naturally dark-colored; as, for instance, at the parts of generation in either sex, where it appears in the form of white spots of various size, that gradually spread, and at last, in some few cases, amount to a general discoloring.

Yellow, either pure or mixed with green, is the well-known color in cases of icterus. A similar hue, but inclining to brown, arises from the deposit of pigment in the epidermis, either in small stains, or in large discolored tracts, or even over the whole surface of the body: the cause of this deposit is still partially obscure. The uniform embrowning of the skin, brought on in parts that are exposed especially to the light of the sun, is of this kind also; as well as the spotted stains of summer freckles (ephelis); and the liver spots (chloasma) which depend upon anomalies in the biliary system, and in the sexual system of the female. The color of the skin generally becomes dark, when with neglect of it and indulgence in alcohol are combined infiltration of the liver with fat, and a tallowy state of the adipose substance, particularly of the subcutaneous layer of fat. The skin, in the last case, feels fatty, soft, and velvety, like that of a negro; its color proceeds from the deposition of a pigment containing fat in the deepest layer of the epidermis,—a fact of particular interest, on account of the combination, just mentioned, in which it stands.

Red coloring of the skin appears in extremely numerous forms, and with various shades of yellowish, bluish, livid, coppery, brown, and so on, which are well known as pathognomonic of various diseases. It occurs in cases of mere congestion, in inflammation, in exanthematous and impetiginous processes, in teleangiectasis and many of the diseases of the textures of the skin. The redness inclines to blue, and even to black, in hemorrhages into the cutaneous tissue, or upon its surface, in sugillations, ecchymosis, vibices, petechiæ, &c.

In cases of cyanosis there is a general bluish or *blue* color of the integuments; but it is principally marked in situations where the skin is delicate and highly vascular, and in the extremities. The blue tint, when limited to certain spots, is a result of local congestion. A transient blueness of the skin has also been noticed, in a few cases, at various parts of the surface, but its internal cause is unknown (Otto).

Spots (livores) of a bluish-red, a livid, or a blackish-blue color, appear upon the body soon after death.

Various shades of *bronze* are produced upon the skin by the long-continued use of nitrate of silver: they sometimes gradually disappear, but occasionally they remain permanently. No evidence has yet been obtained as to the seat of this discoloring: it appears first, and has its deepest hue, on parts of the body which are exposed to light.

A *black* color is observed principally in old cachectic persons, in whom it is sometimes diffuse and extends over large tracts of skin, especially in the lower limbs, and sometimes appears in the form of black nodules, which are deposited chiefly on the face. It has in a few cases been seen gradually spreading over the whole body. It is named melasma, and is a different affection from cancer melanodes of the skin.

Almost all these discolorings occur also, as congenital and partial appearances, in the various nævi.

A *tawny* color, a *dirty gray*, a *dirty bluish*, a *leaden* hue is by far the most frequent of all the changes in the color of the skin: it is an expression of dyscrasia, and of faulty chymification, and is found in the course of acute and chronic diseases.

§ 6. *Anomalies of Texture.*

1. *Congestion,—hemorrhage,—anæmia.*—A passive congestion, limited to certain parts of the skin, may be constantly observed on the dead body. It is seen, too, on the whole integuments, as a dark redness, with a blue or black tint, in the course of acute and chronic adynamic diseases, and in most instances of agony: it is very marked in parts of the

body that are at a distance from the heart, and becomes extremely so if there be any mechanical interruption of the circulation.

Congestion in a higher degree gives rise to hemorrhage into the tissue of the skin; sometimes in small circumscribed spots, sometimes in streaks, and sometimes to a large extent; it may take place upon the surface of the corium beneath the epidermis, or in the tissue of the former; and, in the latter case, it is usually associated with hemorrhage into the subcutaneous tissue also. The bloody spots in Werlhof's *morbus maculosus*[1] and in scurvy, the petechiæ in the course of typhus and typhoid fevers, &c., are instances of such hemorrhage. Its occurrence is facilitated by delicacy and susceptibility to injury on the part of the walls of the capillary vessels, and by a tendency to transudation on the part of the blood.

Anæmia of the general integuments is a local part of a universal anæmia, and is always accompanied with collapse and pallor of the skin; the pallor acquires a waxen character when the skin is delicate, and especially if at the same time it be rendered tense by the presence of fat or œdema.

2. *Inflammations.*—Inflammation of the skin (dermatitis) may result from very various external influences, which are but partly known, and be an idiopathic, substantive disease: it also very frequently occurs as a symptomatic and dependent affection,—a reflex of other morbid processes. Regarded in an anatomical point of view, it is found sometimes diffuse, and extending over large tracts of skin; sometimes it is circumscribed, and confined to one or more small spots.

In the first form, the true cutis is the part attacked, and sometimes only its external layer and papillæ—*erythema:* at other times the deeper layer also, that is to say, the whole thickness of corium is affected; and that constitutes *phlegmonous* inflammation.

From it, and particularly from the erythematous form, there are several transitions to the *circumscribed* inflammation of skin. The simplest form of the circumscribed is *furuncular* inflammation.

Allied to this are several of the acute and chronic exanthematous processes.

I proceed now to speak of them in detail.[1]

a. Erythematous inflammation of skin.—Erythematous inflammation, as has been said, is an inflammation of the outermost layer of skin, which contains the papillæ; and it includes not only the slight inflammation produced by external agents, such as the heat of the sun, fire, cold, irritating plasters, trifling injuries, the stings of insects, &c., but also spontaneous inflammations of exanthematous nature, which are essentially connected with other morbid processes such as the various erythemata, erysipelas, scarlatina, measles, intertrigo, &c.

Erythematous inflammations usually run an acute course, but several of them are apt to recur, and to become habitual.

The following are the anatomical characters of the disease. The redness is for the most part bright and uniform, but sometimes it is irregular, presenting here and there various forms and outlines of a deeper hue, and very frequently it has a shade of yellow: the color gradually diminishes towards the border of the inflammation, and passes imperceptibly nto that of health: it disappears upon pressure, and quickly returns when the pressure is remitted. There is mostly but trifling swelling, uch as can be perceived only by the touch, and at the border of the diseased spot. The exudation is determined by the intensity of the process: sometimes there is none; sometimes a watery fluid, effused slowly or very rapidly, raises the epidermis in small and scattered, or in conluent, vesicles. The under surface of the skin is reddened, has a granular or uneven glandlike appearance, and is covered more or less distinctly with a grayish-white, soft, gelatinous, plastic exudation, which is sometimes perforated and cribriform, and sometimes is reticulated on the surface next the skin. If the inflammation still increase after this product is deposited, the redness becomes darker and the exudation reddish, milky, and at last purulent.

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[1] [*Vide* Behrens' Dissert. Epistol. de Affectionibus a Comestis Mytulis. Hanover, 1735, p. 3.—Ed]

After death the redness has generally disappeared, but the swelling s still perceptible; the epidermis is either easily separable or actually separated, and the surface of the cutis is moist, and covered with a viscid and more or less puriform exudation. The redness is seen on a transverse section to be confined to the outermost thin layer of the cutis; the deeper layer is pale, and is somewhat infiltrated only when the inflammation is intense; the subcutaneous cellular tissue is then in ike manner slightly infiltrated.

Erythematous inflammation generally terminates by resolution, the epidermis peeling off one or more times, according to the severity of the nflammation, in the form of a mealy powder, or a bran-like scurf, or of larger scales and laminæ, until the skin, covered with a new thin epidermis, looks smooth and shining and of its healthy color.

b. Phlegmonous inflammations of skin. (True dermatitis.)—Phlegmonous inflammation extends beyond the papillæ into the deeper strata of the corium, and sometimes involves not only the entire thickness of that part, but also more or less of the subcutaneous cellular and adipose tissue. It arises very often from the contact of powerful external applications, like burning or cauterizing bodies, with the skin; and sometimes, without manifest external occasion, from an internal cause; sometimes, again, it is produced by the extension of inflammation from subjacent structures, from cellular tissue, muscles, veins, or absorbents.

Just as, under certain circumstances, the phlegmonous arises out of the erythematous inflammation, so also has it several degrees of its own, which pass perceptibly into one another. Its course is in most cases acute, but it is often chronic, and then usually becomes acute from time to time.

The following are the anatomical characters of *acute* phlegmon of the skin. The redness of the inflamed spot is generally deep (saturated) and dark, it varies in its tint according to the state of the blood, and does not disappear upon pressure; the swelling is moderate, but the firmness of the skin amounts to decided hardness: the tissue of the cutis is found upon section to be red, and to have a homogeneous fleshy appearance; its reticular structure has disappeared; the fat contained in it has lost its characters; and it is also easily torn: the subcutaneous cellular and adipose tissue is minutely injected, and infiltered with a serous fluid. The under surface of the skin presents more or less redness, and a shreddy, granular appearance, and is covered with a viscid exudation, that as it softens becomes purulent.

Chronic phlegmonous inflammation of the skin, such as is developed gradually out of repeated attacks of erythema, and is kept up by various constitutional affections that arise from the suppression of normal or of anomalous excretions, presents very different characters according to the degree of the inflammation, and the circumstances by which it is occasioned and maintained.

The redness is usually dull, and inclines to a bluish, brown, or bronze color.

The tumefaction of the cutis itself is slight: its density is sometimes increased, sometimes decidedly diminished; and it is accordingly firmer than natural, or loosened in texture, and spongy.

The cutis when exposed is sometimes found smooth, uniformly softened and spongy, sometimes it is unevenly granular, and either soft or rather hard. It is covered with a limpid, watery, and colorless, or with a yellowish, yellowish-red, and bloody, or with a thick, viscous, clear or turbid, and yellowish-white, or a yellow purulent, moisture: these products soon change to dirty white, asbestos-like, epidermal scales, and then peel off; or, becoming thickened and dried, form a covering like various kinds of bark.

The subcutaneous cellular tissue is sometimes infiltrated with a viscid serous fluid, and injected; sometimes it is denser than natural, hard and lardaceous, &c.; as the fat disappears, the inflamed spot is depressed beneath the surface of the adjoining healthy skin.

This inflammation terminates in various ways.

Acute phlegmonous inflammation sometimes terminates in *resolution*, but it leaves the diseased part of a bluish-red color, very susceptible of external influences, and liable to a recurrence of the inflammation for a long time afterwards. It often gives rise to destructive *suppuration* of the superficial layer, or of the whole thickness, of the corium. That coat is then replaced by a cicatrix, which becomes more or less fixed to, and blended with, the subjacent structures: if inflamed and suppurating surfaces of skin be brought into mutual close contact, they may even unite with one another.

Extensive phlegmonous inflammations and suppurations of the skin, particularly those which are produced by burns and scalds, very often lead to a fatal result, either speedily by *exhaustion* of the vital powers during the violence of the fever, or more slowly by their drain upon the blood, by congestions, inflammations of internal organs, especially of the lungs (hypostasis), or by exhaustive serous exudations, particularly on the mucous membrane of the intestines. Burns, and especially, as I have observed, burns of the skin of the abdomen, are in a few instances attended by fatal hemorrhage from the bowels, which is most probably introduced by an exudative process.

Now and then, acute phlegmonous inflammation of the skin terminates in *mortification:* but of this hereafter.

The consequences of chronic cutaneous phlegmon do not cease with the permanent anomalies to which it gives rise in the stratum beneath; these are in themselves of a serious character, but they obtain greater importance from their relation to the integrity of the whole organism. At one time it leaves behind it a *condensation* and *thickening* of the skin, in which the subcutaneous cellular tissue also is generally involved, —hypertrophy with induration, and adhesion of the skin to the subjacent structures.

Under certain etiological circumstances, the inflamed spot becomes a vicarious organ of secretion. The exposed skin, having thrown out a few granulations, secretes a thin fluid, which is often very acrid and corrosive, and gradually eats away the substance of the cutis. If at length, the conditions being favorable, the secretion should subside, and the part heal, the cutis is replaced by a dirty brown, vascular, very vulnerable and frail stratum, which, for the most part, produces large scales of epidermis in considerable quantity and is very long in turning pale, and in acquiring the firmness of a sound cicatrix tissue. The whole metamorphoses is a secondary *atrophy* of the cutis occasioned by the inflammation.

Chronic inflammation of the skin frequently ends in *ulceration*, and especially is this the case when, from some internal or external cause, the chronic is rapidly exaggerated to a more intense degree of inflammation, or when inflammation returns at a part where the change into cicatrix tissue is going on. Suppurative and sanious destruction, in various forms and to different extents, that is to say, ulcers, then ensue with more or less rapidity.

It may happen that several of these terminations of inflammation exist near together, or are associated with a continuance of the inflammation.

c. Furuncular Inflammation.—The forms in which this kind of inflammation occurs are furuncle and anthrax; it occupies the deeper, areolar layer of the corium, and the cellular tissue filling the interspaces of its network. A circumscribed swelling at first presents itself, no larger than a hemp-seed or a pea, which, as it gradually increases in size, becomes remarkable for the (reactionary) inflammation that attends it: for the inflammation forms proportionally a wide halo around the swelling, corresponding with the pain and the marked degree of tension that exist; it reaches also into the deeper structures, and fixes the swelling of the skin to the subcutaneous cellular tissue. Before reaching its highest point of severity, it furnishes a product that is known by the name of a (Pfropf), *core* or plug. This product has been regarded as sloughy cellular tissue; but, upon more thorough examination it is found to be a product of the inflammation going on in the cellular tissue contained in the meshes of the corium, and to resemble false membrane,—to be, therefore, exudation. It occupies the whole thickness of the corium, and exists there before the swelling is very perceptible: at first, it is closely connected with the surrounding injected tissue, but, as the (reactionary) inflammation around it produces suppuration, it is thrown out. The core has in fact nothing in common with separated sloughy cellular tissue, it is exudation; though it certainly may contain a few fibres of cellular tissue interwoven with it, which have been severed from the rest by the suppuration going on around it. (Gendrin, Aschersohn.)

In furuncle only one such product is formed; in anthrax there are several of them near together. The reactionary inflammation around and beneath is very considerable, corresponding in degree to the pain and the feeling of tension. If before the commencement of suppuration, and consequently before the loosening of the cores, an incision be made into an anthrax, a uniformly red, spongy, or reticular tissue is exposed, the meshes of which are filled with cores. At a later period, when the cores are loosening from the inflamed tissue, and suppuration is just coming on, each core is found surrounded by a substance like jelly. When, at length, suppuration is established, the cores become completely separated, and, by the destruction of the meshes of the network, cavities of different sizes are formed, in which they freely swim.

Instead of leading to the production of matter, and suppurative destruction of the tissues, the process sometimes terminates in another way; for, under a combination of excessive local tension, and unfavorable general circumstances, viz., in the condition of the individual, and in external influences, mortification takes place, especially in the skin covering the carbuncle.

Furuncle very rarely terminates in induration; it might, when such is the case, be confounded with several other circumscribed inflammations of the skin.

There are various other inflammations of the skin allied to furuncle, but differing from it: some are primary, others secondary (or, as they are called, metastatic, critical). Some are parenchymatous, others proceed from particular parts of the structure of the skin, especially from the sebaceous glands. They may terminate in suppuration or in induration.

d. Exanthematous Inflammations.—To this class belong all acute and chronic exanthematous processes which present the following general characters. They are preceded or accompanied by symptoms of inflammation: either at one spot, or at several, separate or clustered points, they furnish a product: sometimes that product takes the form of vesicles and bullæ, and lies between the cuticle and cutis; sometimes it occupies the parenchyma of the cutis, being effused amongst the papillæ, or in the deeper layer, and forming nodules that either subside again, or suppurate and produce small abscesses or pustules; and sometimes, lastly, it gives rise to induration of the skin, to nodules and nodular thickening of the subcutaneous cellular tissue, and their usual consequences, suppuration, ulceration, or hardening.

The present appears to be the most convenient opportunity for alluding to these processes; for anatomy has not yet furnished satisfactory evidence as to their real site, whether it be the different glandular organs of the skin and their ducts or not. I venture, however, to omit giving any minute description of them, not for this reason merely, but also because we possess only a few fragments of anatomical information respecting their products; and further because the changes in the internal organs, which have hitherto been observed after death,—changes, that is, essentially connected with the disease of the skin, and constant in their occurrence in many of these cases, though not, indeed, in all of them,—do not furnish facts in sufficient number and of a kind to allow of our constructing an account of their pathological anatomy, that would make any pretensions to truth and to practical utility.

The last remark applies particularly to those exanthematous diseases which are usually treated of amongst chronic diseases of the skin, for several of those which are now under consideration, as well as of the acute processes spoken of under the head of erythematous inflammations, are frequently fatal, and consequently become the subjects of pathological examination, especially true variola and scarlatina.

Although much has already been said, and some remains still to be said on the subject, it will be proper to mention here, in general, some of the principal results of examinations of the body in the cases of exanthema, that have been mentioned.

With an exanthema upon the skin that is discolored, collapsed, and sometimes scarcely perceptible, are connected erythemata and exudative and pustular inflammations upon the several mucous membranes adjoining the external integuments. They take place especially on that of the mouth, pharynx, trachea, and bronchi, as well also as that of the urethra and vagina; they are complementary to the cutaneous eruption, and may be more or less substantive in their character; but frequently they are extremely developed both in extent and in the degree of their intensity.

Next in order may be placed the more or less palpable developments of the follicles of the intestines, especially those of the ileum; after these, similar developments of the mesenteric glands: and then congestions and enlargements of the spleen.

These may be followed by congestions of the central organ of the nervous system, and of its membranous investments; and very commonly, too, by increased density of the cerebral substance, with the exception of cases to be mentioned afterwards.

And next may be mentioned exudative processes upon mucous and serous membranes, especially on the former: some of these are genuine croupy exudations on the divisions of the mucous system above mentioned, as croupy pneumonia, croup in the œsophagus, stomach, and intestines; others are gelatinous, purulent or serous exudations, and are found particularly on the mucous membrane of the bowel, and in the parenchyma of the lungs,—pulmonary œdema; others, again, are exudations upon serous membranes, especially upon the arachnoid, where they are accompanied with a turgid, moist, and loosened state of the cerebral substance,—œdema of the brain; some are met with on the pleuræ, &c., and even upon the internal surface of the vessels (phlebitis).

The acute black softenings of the cardiac portion of the stomach, and of the œsophagus may be arranged next; and then—

Gangrene on the general integuments and in the internal organs.

Changes of the mass of the blood take place in the dead body parallel with these processes in the solids. The fibrin has a marked tendency to coagulate; the blood is deprived of its fibrin; or the latter is fluid; or the blood contains no coagulable part, but is either no thicker than water, or thick, viscid, like tar, and of a purple-red color, inclining to bluish, violet, black, &c. With the changes last described, are connected marked collapse of the body, lividity of the integuments (especially of the exanthematous part), and of the muscles, red transudations into the serous cavities and into the tissues, and particularly the escape of blood into the parenchyma of membranous expansions, in the form of ecchymoses, petechiæ, suffusions, &c., especially on the skin.

As the exanthematous, especially the acute exanthematous processes are allied in their nature to the exudative, I must here refer to yet one septic exudative process which takes place upon skin deprived of its epidermis, and which is closely analogous to sloughing croup[1] (Bretonneau's Dyphtheritis): it is that which is named hospital gangrene.

3. *Ulcerative processes.*—The ulcerative processes are, for the most part, results of inflammations already described; and they are especially liable to occur when those inflammations, having been raised to unusual intensity by some unfavorable external influences, either continue intense or repeatedly become so; or when they are called forth by some internal constitutional cause (dyscrasia); or when running their course under such constitutional influence, they give rise to a special product by which the tissues are in a peculiar manner consumed (dissolved). As the inflammations, especially the various exanthematous forms of inflammations present numerous characters, which more or less distinctly manifest the nature of the constitutional affection, so also, and still more, are these characters usually stamped upon the ulcer.

Again, many ulcerations of the skin are produced by the metamorphosis of known adventitious growths in the skin itself or in the tissues beneath it; others are secondary stages of various changes in the texture of the cutis, with which we are not as yet acquainted.

Of this kind the following are examples, although most of them still require minuter anatomico-physical investigation: All ulcers connected with disorder of normal, or what have become normal excretions: all those which originate in a congenital or hereditary, or in an acquired dyscrasia, whether the latter be simple, or combined and modified: all menstrual, hemorrhoidal, and urinary ulcers, as they are called, are therefore of this kind; so also are the abdominal, the gouty, and the scorbutic ulcers, those which exist in psoriasis, the syphilitic and syphiloid, the leprous, scrofulous (tubercular) and cancerous, and the numerous cancroid ulcers. They present many more or less characteristic differences in site and in form, i. e. in the state of their margins and bases, in their disposition to extend superficially or deeply, and in the amount, and especially in the quality of their product: hence the known divisions of ulcers into round, oval, and sinuous; into callous and fungous; into moist and dry, &c.

As the ulcer presents various characteristic peculiarities, so also does the cicatrix.

It is important and interesting to observe the relation subsisting between inflamed and ulcerating integuments and certain subcutaneous structures, especially periosteum and bone: it is seen, for instance, on the cranium and shins, and prevails chiefly in the inflammation and ulcer arising from constitutional causes.

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[1] [Gangrenous stomatitis?—Ed.]

4. ***Mortification of Skin.***—Mortification is not an unfrequent occurrence in the skin; it arises from congestion and inflammation, and takes place more readily the more insuperable the mechanical interruption to the circulation, and the greater the exhaustion of nervous power either in the system generally, or—in consequence of extreme severity of the inflammation or unfavorable external circumstances—in the part itself. Sometimes it takes the form of moist, sometimes of dry gangrene,—sphacelus—mummification. In the former, the epidermis is raised in vesicles of various size, which are filled with a discolored sanguineo-sanious fluid, and the tissue of the skin degenerates to a loose, pulpy, and offensively smelling mass of a brownish, brownish-green, or blackish color: in the latter the cutaneous tissue changes to a black, pretty firm, dry eschar, which is frequently puffed out with gas, developed in the subcutaneous tissues, when they are affected with moist gangrene.

The mortification may extend from the skin to the subcutaneous tissues or *vice versâ*, or again it may attack both structures, however heterogeneous, together.

Examples of primary gangrene, as well as of the secondary proceeding from inflammation, are furnished by gangrena senilis, by the bluish-red congestion and gangrene that occur, often at several spots, on paralyzed limbs, by the sphacelus accompanying or following typhus and typhoid fevers, by bed-sloughs, anthrax (pustula maligna), cancrum oris (noma), by the mortification that takes place from various internal or external causes in inflamed skin, in ulcers, and in wounds, and by hospital gangrene.

Besides these two forms of mortification, a third is sometimes observed, which has been termed white gangrene (Mayo, Aschersohn). The skin becomes converted into a dirty yellowish-white, or grayish-white, friable eschar. A similar form of necrosis of cellular tissue has been pointed out already, and a similar eschar on serous and mucous membranes. It may arise from the stretching—which is equivalent to compression—of the tissue, or rather of the capillary vessels by an inflammatory swelling, or from the vessels being destroyed in necrosis of the subcutaneous cellular tissue (pseudoerysipelas).

5. *Adventitious Growths.*—Here again I cannot avoid remarking that, whether from the apparent insignificance of these growths in themselves, or from the regard paid to that affection of the internal organs which gives occasion for the examination of the body, the most accessible of them have received as yet but casual anatomical notice, and the distinctions between them have continued based upon their external characters only.

a. New growths of *cellular tissue* occur as:

α. Soft wartlike growths, attached by a pedicle, which constitute what is called the *Molluscum simplex:* the saccular dilatations of the corium are occupied by some cellular tissue at various stages of its development. They occasionally also contain fat.

β. ***Fleshy excrescences*** on the nose—exuberant or bottlenose: these are composed of a luxuriant growth of corium and of cellular tissue.

γ. *Condylomata* are common about the organs of generation and the anus, especially on the mucous membrane of the former. Some of them are soft, others firm: in their form they may be broad and pointed: sometimes they are attached by a pedicle, and very often their extremities resemble a mulberry, a cauliflower, or a cock's comb. They are composed of an investing layer of epithelium and of newly formed cellular tissue; and they originate in the corium, where their points, which, as is well known, are the more unmanageable part, take deep root (Simon). With these most probably we may connect those out-growing tumors which occur in the Pian of tropical climates, and have by many been regarded as syphiloid; as well as various affections that are met with on the coasts of Europe, for instance, the Radesyge, &c.

b. Fatty tumors are usually congenital: sometimes only one exists; at other times there are several, which are situated at other parts of the body. They form rounded, globular excrescences, which are, for the most part, truncated and attached by a pedicle, and sometimes grow to a considerable size. They consist of a prolongation of cutis, and enclose some fatty tissue, which seems like a protruded lobule of subcutaneous fat; for at the base or neck of the excrescence it is continuous by a sort of pedicle with the general subcutaneous adipose stratum. The epidermis covering them is sometimes dark-colored, and pigment (Nævus lipomatodes of Walther), and unnatural hair often grows upon them. When it is a congenital disease, it is often associated with nævus in other parts of the skin. In some few cases, these lipomatous growths are developed in later periods of life.

c. Fibroid tissue occurs in skin thickened by repeated, or by chronic, attacks of erythema; in the wheals and knolls of the skin in cases of elephantiasis, &c. It also constitutes cicatrix tissue.

d. The growth which Alibert has denominated *cheloid* may probably be placed in connection with the last named; for it appears to consist of fibroid callus, and with that appearance its external cicatrix-like aspect corresponds. There are several varieties of cheloid; it may be a simple hardness or callosity of the skin, either flat, somewhat raised, or depressed, and white or pale rosy-colored; or it may be cord-like: in either case it frequently terminates in white or red elevated lines or processes (the *spider-like pimple* of Warren), and is of considerable extent. It occurs, for the most part, singly at the upper part of the trunk, on the extremities, or on the face; in very few instances does any large number exist.

It very rarely ulcerates; when it does so, the sore may now and then have a malignant (bösartig) character. Some constitutional disorder lies at the root of every case, but the nature of it is unknown; that it is cancerous is altogether problematical.

e. Anomalous bony substance is extremely rare in the skin. I once found, in the substance of a scar on the trunk, an oval, yellowish, hard, rugged, osteoid plate, about the size of a thaler.[1] It corresponds precisely with the calcareous growths occurring in fibroid exudations upon serous membranes.

f. Teleangiectasis in skin is the well-known vascular nævus; it is almost always congenital. Sometimes it forms deep-red, or bluish-red, stains of extremely various size and form (Feuermal,—moles), and sometimes red tumors, which are shaped like cherries, strawberries, mulberries, &c., and are capable, more distinctly than the former kind, of a transient swelling (Dupuytren's erectile tumors,—splenoid tumors of other authors). But they do also commence in after-life, and in themselves are at first quite of a benignant, that is to say, not of a cancerous, nature. Nevertheless, if there be a cancerous cachexia, the teleangiectasis may unquestionably become the seat of a cancerous growth; and, under such circumstances, it has been taken for a special form of cancer—Fungus hæmatodes cutis: it is not, however, an essential form of that disease, but is merely an accidental complication.

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[1] [About the size of an English half crown.—Ed.]

Teleangiectasis consists of a network of enlarged capillary vessels, imbedded in a delicate, and partly undeveloped cellular tissue.

g. Melasma, (benignant) melanosis includes both the black coloring which is observed, in some few cases, spread over the whole body, but more frequently limited to certain parts, especially the lower extremities; and also more particularly the accumulation of deep black pigment, in small raised points and berry-like tumors on the trunk and face. The pigment is deposited on the surface of the cutis, and in the latter case in its tissue also. Melasma occurs only in aged, decrepit, and cachectic persons, and must, of course, not be confounded with cancer melanodes.

h. Cysts.—Newly formed cysts do not occur in the skin itself; but, instead of them, the sebaceous glands not unfrequently degenerate into cysts of large size, of which I shall speak presently. There are often also cysts in the subcutaneous cellular tissue which become, in various ways, closely connected with the corium. Such cysts very commonly contain cholesterine, which is also quite constant in the morbidly enlarged sebaceous follicles.

i. Associated with these is the occurrence of *cholesteatoma*, as a stratum covering open ulcers of the skin. I have not only met with this upon carcinomatous ulcers, as others have observed it, but have also seen it produced exuberantly, in large masses, on an ulcer of the skin of the right knee, arising from burn.

j. Tubercle.—There appears to be no tuberculosis of the skin corresponding to that which occurs in and upon mucous and serous membranes, parenchymatous structures, &c.; at least its existence is altogether problematical. Upon ulcers, however, which arise from various exanthematic cutaneous affections of what is called scrofulous character, a product resembling softening tubercle, or puriform tubercular matter is seen, from which the ulcer obtains a character approaching that of the ordidary tubercular ulcer upon mucous membrane, particularly the intestinal ulcer.

The skin is subject, also, to ulcerative softening of a less definitely tubercular character during the softening of tuberculous lymphatic glands, and of tubercular depositions in the subcutaneous cellular tissue, as well as when there is ulcerating tubercle in bone.

k. Cancer, and cancerous ulcer, are of frequent occurrence in the skin. Cancerous degeneration and ulceration of subcutaneous tissues very frequently involve the skin over them, and cancers of the subcutaneous cellular tissue, and of glands imbedded in it, particularly of the mammæ and lymphatic glands, usually become at an early period very closely connected with the cutis. But cancer also originates in the skin, presenting itself, according to my observations, under the forms of fibrous (scirrhous), and of medullary cancer.

a. The form which fibrous cancer assumes in the skin is that of a rounded, or rounded and tuberculated nodule; very often it is flattened, or even depressed beneath the surface of the skin, and then it lies in a sort of umbilical fossa. It is generally single, about the size of a hempseed, pea, or hazel-nut, firmly fixed, and as hard as cartilage: sometimes it is smooth and shining externally, and sometimes covered with a hard laminated crust of epidermis; and frequently it is somewhat darker than the skin around it. When examined closely, the outer strata of the nodule are occasionally found transparent. It occurs principally on the face, lips, and nose, but is occasionally found on other parts of the body: it is generally the primary cancerous growth, the first of a series of cancerous formations in different organs of the body. In some few cases it reaches a considerable size, growing out into a tuberous mass that projects beyond the skin.

β. Unlike fibrous cancer, the medullary kind is usually a secondary formation, and associated with large cancerous growths, which first appear just beneath the skin, or if they come from a greater distance involve the subcutaneous structures first, and then the skin itself: in either case it grows in the skin in isolated or confluent nodules near the primary mass. At other times it comes on in skin after cancer has been already localized in one, and still more when it exists in several organs; it then constitutes one part of an extensive, or it may be, of a general, production of cancer. The nodules which it forms are mostly numerous, and about the size of peas or hazel-nuts; they are scattered over large tracts of the body, especially over the trunk, and near similar growths in the subcutaneous cellular tissue. It is distinguished in the skin, as well as in that tissue, as a whitish or whitish-red growth, which is sometimes tolerably firm and lardaceo-medullary, and sometimes softer, looser, and resembling cerebral substance, or even diffluent like milk, and which grows to considerable size. It frequently corresponds with a character which may be possessed by the fundamental or primary growth, in also containing pigment; and in that case it constitutes cancer melanodes of the skin. The layer of skin which at first existed above the medullary nodule, becomes stretched, and sometimes is shining and transparent, sometimes rough from having lost its covering of epidermis: at a later period, as it is being perforated, it becomes moist, and furnishes the nodule with a cortical covering; and a remarkable villus-like development of its papillæ takes place, which appears, from the result of observations upon chimney-sweeper's cancer, to occur in an especial degree in that disease. Sometimes the elementary particles of the disease are deposited in a pre-existing teleangiectasis, or, as the deposition takes place, there happens an excessive development of the vessels of the skin: the result, especially in the former case, is a cancerous structure of uncommon vascularity, which then receives the name of *fungus hæmatodes.*

Chimney-sweeper's cancer, and Alibert's eburnated cancer of the skin, must be referred to as special varieties of the disease.

Chimney-sweeper's cancer appears to be of medullary nature. It almost always begins, as is well known, on the scrotum with a tolerably firm, small nodule, or a warty excrescence, which, after having existed for some time, becomes red, excoriated, moist, and covered with a cortex: the papillæ beneath it enlarge considerably, and at length the whole becomes an ulcer with irregular, hard, raised edges. Fresh nodules form around it during its progress, and, by the ulceration of these, the original sore enlarges, for the most part superficially: the nodules at the same time become developed into fungous cauliflower excrescences, and at last the metamorphosis extends deeply. After infiltration and induration have taken place in the dartos and tunica vaginalis, and the latter has become adherent to the testicle, that gland itself ulcerates, while the adjoining lymphatic glands and the vas deferens degenerate quite up to the abdominal cavity.

The *eburnated cutaneous cancer* of Alibert is a diffused degeneration which occurs, without doubt, only as a secondary affection, the skin being destroyed in the degeneration of cancerous growths beneath it, at an advanced stage of the cancerous dyscrasia. Over a scirrhous subcutaneous cellular tissue, the cutis is stiff and immovable, white, glistening, and somewhat transparent, and the whole mass is uncommonly firm. Although the disease is very rarely observed with so marked a character as Alibert has seen, yet now and then an opportunity occurs of examining cancerous degenerations of the skin, which in some degree approach what Alibert has described as *carcine éburnée.* It is quite uncertain, from its elementary structure, to what form of cancer it belongs, but, from the state of the disease with which it is connected, it should be the fibrous form.

From any of these cancerous growths a cancerous ulcer may be formed. Congestion and inflammation come on in and around the growth; and, while it becomes turgid, dark-colored, and vascular, and a fungous growth protrudes, it softens and splits, and, producing a cancerous sanies, breaks down. At the same time, new cancerous matter is deposited, either by infiltration or in nodules, in the tissue forming the margins and base of the principal ulcer. This metamorphosis of the cancer, as well as the softening of the secondary deposition in the ulcer, may run its course either with or without a fungous protrusion. The former is particularly characteristic of the ulcer of medullary disease; while, on the contrary, there are some remarkable cancerous ulcers, by which tissues are eroded not only without visible previous cancerous degeneration, but even without any considerable production of sanies. Ulcers of this kind do unquestionably often originate with one of the cancerous growths already mentioned; though not always, for they are sometimes developed secondarily from some injury or ulceration. They frequently produce extensive devastations, especially upon the face, and commonly attack and destroy all structures without distinction; for which reasons, as well as from their ungovernable nature, they are regarded as cancerous; but varied and accurate investigations of all their characters are still required.

l. Parasites.—Several kinds of pediculus, the itch insect (acarus scabiei), and without doubt other acari also, occur both in and upon the skin: the subcutaneous cellular tissue is infested with the filaria medinensis already alluded to (p. 22).

Among vegetable productions may be mentioned the thread fungi (fadenpilze), which are formed in the pustules in cases of porrigo favosa. They are the primary anomaly, and constitute unquestionably the essence of the whole disease.

Appendix.—Anomalies and Diseases of the Sudoriparous and Sebaceous Glands.

A. In several of the exanthematous processes the sudoriparous glands and their ducts are unquestionably subject to frequent and various diseases, both primary and secondary, but the anatomical investigation of their diseases is attended with many difficulties, and no advance has yet been made in it. Our knowledge is limited to the anomalies in the quantity and physical properties, most of them, therefore, symptomatic anomalies, of their secretion, *i. e.* of the perspiration: but chemistry has hitherto supplied information in some striking cases only, and the investigation is beset with as many hindrances as before.

B. The sebaceous follicles and their excretory ducts are certainly the true and the original seat of many exanthematous processes; but their most frequent morbid condition is enlargement, arising from the accumulation of thickened secretion within them. The least degree of the affection, and a very common one, is dilatation of the duct of the gland, and is known by the name of *Mitesser*,—maggots (*Comedones*). The accumulation of the secretion in the sudoriparous sac itself produces white rounded tumors,of the size of gravel, or millet-seed. When dilated to a greater, the sac degenerates, either alone or together with its excretory duct, into a cyst as large as a pea or a hazel-nut, or even larger; when it is diseased alone it is opened externally; but in the latter case it separates from its duct, and completely closes: it contains a whitish, laminated, firm substance, like adipocire, or a pulpy substance, viscid like fat, and consisting of strata of epidermis, and crystallized fat. In all these forms the disease occurs principally in the larger sebaceous follicles on the face, at the upper part of the trunk, on the back, and in the neighborhood of the parts of generation.

The diseased sebaceous glands frequently give rise to inflammation of the adjoining corium—to acne,—an inflammation that sometimes goes on to suppuration of the follicle, as well as frequently of the bulb of the hair with which it is connected, and sometimes to induration (acne indurata), and thereby to a slow cure.

In large sebaceous cysts the epidermal mass sometimes takes the form of a horny excrescence,—a growth to which I shall advert presently. In other cases their contents become inspissated, and form calcareous concretions.

The occurrence of a condyloma in the sac of one of these glands—*condyloma subcutaneum* of Hank—is a very interesting phenomenon, which for that reason requires further investigation.

The secretion of the gland is sometimes more abundant than natural, and is poured out upon the surface of the skin (seborrhagia): it dries there in thin whitish, glistening laminæ, or in thicker, dirty strata or scabs, which feel like fat.

Anomalies and Diseases of the horny tissues,—the Cuticle, Nails, and Hair.

A. The *Cuticle* is subject to several anomalies, but they are not accurately known; and their relations to diseases of the cutis require especially to be explained.

§ 1. It is very often formed in excess; and then either its outer layers are thrown off in the form of bran, scales, larger coherent masses, &c.; or its elementary structures, accumulating upon and beside one another, produce very various secondary formations, such as callosities, corns, and crusts, flat, convex, or concave scutes, cylindrical or angular, tessellated growths, and others which resemble stalks and thorns. Anomalies of this kind may be limited to certain circumscribed spots, or may extend over the whole body.

On the other hand, the cuticle is sometimes remarkably thin and delicate, and, therefore, transparent, at spots where it has been recently cast off.

§ 2. An unnatural aggregation of the elementary constituents of the cuticle, and a simultaneous excess of its growth, produce the anomalies in the form of the epidermal tunic which have been already mentioned. I shall refer to this again amongst the anomalies of its structure.

§ 3. Anomalies in the color of the skin reside for the most part in the epidermis. Its cells contain a pigment, perceptible chiefly in the deeper layers, which varies in quantity, and may be yellow, brown, or black. Such varieties in its color constitute the distinctive peculiarities of certain individuals, and certain races, but sometimes they are acquired. In the latter case the change may be limited to particular spots, or may extend over the whole body; and it presents considerable interest from its involving not only marked alterations in the condition of the organ, and in its secretion, but also anomalies of internal organs which indicate a revolution of the entire vegetative system. Pigment accumulates and discolors the skin in a remarkable manner in congenital nævi.

Total absence of pigment is a congenital defect in cases of Albinoismus, and an acquired in cases of Achroma or Vitiligo. The former may be general or partial; the latter is at first always partial, but may at last become general.

§ 4. The epidermis deviates from its normal consistence in being sometimes more or less moist, but more commonly very dry and harsh. It is the latter condition that produces its tendency to break and peel off in the form of bran or scales, as it is observed to do in many substantive diseases of the skin, and in cases in which it is a symptomatic occurrence and the skin is destroyed, especially by cancer. In those cases, likewise in which epidermis has accumulated in a thick layer over a diseased spot of skin, its dry condition occasions cracks, fissures (chaps, rhagades), which not unfrequently extend through its entire thickness, and even into the cutis.

§ 5. The mutual relations, as to position, which subsist naturally between the elementary structures of the epidermis, are frequently disturbed, not only in consequence of their simple accumulation, but also by a simultaneous excessive development of the papillæ of the cutis, and by various other accidental circumstances. Such anomalous relations of structure may be reduced generally to the two forms of a more developed laminated arrangement, and an apparently fibrillated structure. This class includes the anomalies in the shape of the epidermal tunic already mentioned,—

The *callosity*,—tyloma,—which consists of a simple accumulation of epidermis in the form of strata lying over one another:

The *corn*,—clavus,—a small circumscribed painful callus that projects like a wedge into the corium;

The *crusts*, and the convex, flat or hollowed (concave) *scutes* exhibit a laminated structure, though the granular accumulations of dried exudation and pus frequently render it indistinct: but the cylindrical and angular formations resembling pavement, stalks, and thorns, though they also consist of lamellæ of epidermis, are fibrillated, and the horny excrescences have a similar structure. The cutis upon which they grow is always diseased, though not, indeed, to the same degree in all cases; but beneath such growths it is unusually succulent, loosened, vascular, and hypertrophied, and is developed, especially at its superficial lamina, into mushroom-like, cuneiform, thready, villous or even cleft, papillæ. It is evidently so in genuine ichthyosis, and very probably in the milder allied forms of pitiriasis, psoriasis, and lichen. The primary and the secondary changes in the tissue of the skin in lepra are alike unknown.

Horns—cornea cutanea—either grow upon a cutis, diseased in the way just described, or spring from its deeper part, out of a cyst, which is, in fact, a degenerate sebaceous follicle. They have been met with at various parts of the body, but their principal site is the hairy part of the head, and the forehead: they occur sometimes even on the prepuce and glans penis. Usually only one exists, but sometimes there are two or more. Their length is occasionally very considerable, even as great as several inches; and they may be as thick as a finger: some of them are straight, others are twisted or curved; most of them are single, a few are cloven; their broadest part is always the base, the shaft is cylindrical or obtuse-angled, and the free extremity is generally pointed. As to color, they are mostly dirty brownish or black. They have been several times observed to be repeatedly shed at regular intervals. And when thus shed, or when accidentally or designedly removed, they are reproduced, provided the spot of skin which they spring from—the matrix—be not destroyed. They are somewhat more frequent in the female than in the male sex, and are common to old age rather than to other periods of life.

B. The *Nails*.

§ 1. These have in some few cases been found wanting in all, or in some of, the fingers and toes. They are very frequently absent in ill-developed supernumerary fingers and toes. When such parts coalesce, the corresponding nails unite into one plate.

There are various forms in which a preternatural number of nails exists: not only are they in excess when there are supernumerary well-developed fingers and toes, but even when a duplication of the last phalanx is but just indicated; and even without having any trace of being double, a finger or a toe sometimes has a double nail. After the loss of the terminal phalanx a new nail is sometimes formed over the second or first joint, or even upon the knuckle.

§ 2. The size and form of the nails are subject to several congenital and acquired anomalies. Those of the latter class are particularly frequent and interesting; for from them arise the excessive growth, combined with thickening and deformity, which the nails sometimes present in a striking degree. They reach a length of several inches, become misshapen and thick, and twisted and curved like horns and talons. The principal cause of this deformity of the nails is a want of care of them, especially when to that is added neglect or inability to employ the limbs. Of other causes we are ignorant;—a remark which applies particularly to those cases in which an exuberant growth of the nails concurs with the development of horns, &c.

On the other hand, the growth of the nails is arrested in paralyzed limbs, and during the repair of fractures (Günther). They shrink too, and at the same time may be deformed or may retain their shape. When the phalangeal bones waste, the nails merely diminish in a corresponding degree; but simultaneous diminution and deformity is a state, the cause of which, and its connection with other diseases that sometimes coexist with or precede it, are still matters of conjecture. Both in old and in young persons some of the nails, and at last all of them, may lose their smoothness and polish, and cease to grow in length, while they increase in thickness. They then become dry, and split; first the upper shorter lamellæ are thrown off, and at last the whole nail; and the subjacent matrix gradually assumes the nature of the other general integuments, and never produces a new nail. Sometimes the disease is limited, not indeed permanently, but yet for a considerable time, to a state in which small rugged stumps of the nail remain, together with its root: in other cases the nail withers at its lateral margins first, separates from the skin beneath, becomes everted, &c.

Again, the nails present several anomalies in respect to their form, amongst which the convex shape, in cases of extreme cyanosis, is particularly marked. In lepra, in plica polonica, in syphilis, &c., the nails are, in various ways, deformed; and newly formed nails, as is well known, are usually at first misshapen.

§ 3. The consistence and texture of the nails are frequently altered, sometimes independently of disease of the epidermis, sometimes in consequence of it. In the above-mentioned diseases of the skin and hair more especially, they become loose, soft, succulent, and at the same time discolored, or on the contrary dry, as brittle as glass, fissured, &c.

The corium which surrounds the nail is often the seat of an acute, or of a chronic inflammation, which usually ends in suppuration and loss of the nail; and in the toes, when the nail *grows in*, as it is called, is now and then attended with a very considerable growth of granulations (paronychia). In some individuals the inflammation terminates in an ulceration which has the specific character of scrofula or syphilis.

C. The *Hair* presents various anomalies:

§ 1. It may be congenitally deficient in whole or in part (Alopecia connata): the deformity which is thus produced lasts only so long as the growth of the hair is delayed; sometimes, however, it continues throughout life. Partial deficiency involves in some cases all the hair of the head, in others all that in the pubic region; or it may affect small circumscribed spots which remain bald. Allied to this is another condition in which the hair grows sparingly.

Deficiency of the hair is more commonly acquired (Alopecia, Calvities), and may be transient, the lost hair being replaced by new, or it may prove a permanent defect. When the gray hair of the aged person falls out, it is permanently lost; in younger persons the loss is not preceded by any change of color, and is often limited to certain circular spots, which become bald, and increase in extent, though there be no disease in the growth of the hair. Moreover, the hair is sometimes permanently lost in consequence of various diseases and disorganizations of parts of the skin which are naturally covered with it, or in consequence of general weakness and cachectic states of the system, such as syphilis, &c.

But again, there sometimes occurs an exuberant growth of hair; it may be very thick (numerous) at the parts where it is usually found, and grow to an uncommon length; or it may present itself in an unusual situation; or it may appear at an unusual time, either coming forward prematurely or growing anew in advanced life. Thus the hair of the head is sometimes uncommonly thick and long in women, and that of the beard in men; and in the former sex particularly, that on the pubes sometimes presents the same peculiarity. Occasionally the whole body is covered with hair (hirsuties), and sometimes particular parts of it, as the shoulders, the back, the abdomen (Osiander met with a case in which hair began to grow above the navel in a pregnant woman), or the lower limbs in both sexes: sometimes a beard grows in women: in cases of hemicephalus, the hair reaches down to the eyebrows and root of the nose; a long streak of it is found on either side of the spina bifida; hair exists also on nævi, &c. Sometimes children are born with an unusual quantity of hair; or again the hair appears on the pubes at an early age, the hair of old persons grows again, &c. Moreover, the hair is sometimes exuberant at certain parts, while at others it is thin; thus the beard is occasionally strongly grown in persons who have always had a scanty covering of hair upon the head, or from whom it has prematurely fallen off.

Here, too, may be mentioned the occurrence of hair upon mucous membranes, and on the inner surface of encysted tumors. In the latter case, it is almost invariably accompanied by a growth of fat, and very frequently of teeth;—a peculiarity which reminds us of the concurrent deficiency of both teeth and hair, which is sometimes observed (Danz), and of the renewed dentition which is associated in the aged with a new growth of hair. Cysts of this kind are most common in the female sex, especially in the ovaries. Most of the hair, or all of it, lies loose, and steeped in the fat which is present with it, or rolled up in coils within the cysts: plates resembling cutis, however, are sometimes seen on the inner surface of the cyst, and upon them the pores are apparent out of which the hair has grown, or in which it is still inserted. Its development in that situation is similar to that of the natural hair.

Lastly, microscopic hair has been discovered in anomalous secretions of the skin and of various mucous membranes.

§ 2. The hair sometimes presents an excessive growth in length and thickness, especially the hair of the head, where the thickness of the shaft is sometimes considerable. Usually the hair is not only hypertrophied but closely set, and in women the growth may be so abundant as to diminish the *embonpoint* of the body.

It more frequently happens that, in consequence of atrophy of its matrix, the hair becomes thin: it ceases to grow, and becomes somewhat lighter colored than usual, and dry, at last it falls out, and never grows again. This atrophy may be idiopathic, or it may be secondary, and occasioned by various diseases of the skin.

Moreover, there are sometimes found amongst the natural hair, some which are unusually thick, stiff, and brush-like. Albinoes, on the other hand, originally have uncommonly thin soft hair, like the Lanugo.

§ 3. An interesting anomaly in the form of the hair is that in which it bends or breaks, and swells at the broken part into a kind of knot; or, again that in which it splits at its free extremity, and looks like a brush. In a few cases curly hair has usurped the place of smooth; and in one case the hair of the head became curly during an attack of gout in the head. Peculiarities of this kind depend on the state of the medullary contents of the cylinder of the hair.

§ 4. Its color is subject to many changes, some of which are observed only in extremely rare cases. The soft, thin hair of albinoes is white, and has a silky gloss. In Achroma the hair becomes colorless upon spots of skin that have lost their color; but a far more frequent instance of acquired discoloring is its change to gray without previous or simultaneous alteration of the skin. It is chiefly observed in aged persons, in whom it gradually extends from the hair on the head to that on the rest of the body; when it occurs in young persons, it is generally limited to the head. Small circumscribed spots of gray hair are occasionally seen in childhood and youth, and this lack of color is generally persistent; under certain circumstances, however, hair that has turned gray becomes colored again. But not only is it exposed to a gradual loss of its early hue as old age advances, it sometime also changes suddenly to gray, in consequence of extremely depressing affections of the mind.

Still more uncommon are the cases in which the hair assumes a deeper shade than natural, or undergoes an actual change of color. Paroxysms of certain diseases, of gout, of quartan ague, and even the period of ordinary pregnancy, have been stopped by it. Isouard relates, that the blond hair of a woman whom he observed, turned somewhat red as often as she had fever, &c.

§ 5. Faults in the consistence and texture of the hair sometimes present themselves as unnatural dryness, and occasion it to break and split, and, at last, to fall out; at other times, as a morbidly moist and loose condition. The latter results from the deposition of a viscid purulent matter in the pore, and is seen in cases of favus, but is far more distinct, and reaches its extreme in elf-lock (Plica polonica,—Cirrhagra). The hair capsule or pore in this case is tumid, injected, and filled with a pasty, opaque fluid, while the hair itself, from the root upward, is thickened, soft, and full of moisture, and its canal is dilated. It grows rapidly during life; the product just mentioned is effused at its roots upon the surface of the skin, and trickles also out of the hair itself: in this manner the hair on the head, beard, or pubes, sticks together in inextricable tangles, which resemble in shape queues, wreaths, caps, &c. This genuine plica polonica must be distinguished from the similar tangling which takes place in the course of acute, and especially of typhous diseases.

PART VIII.

ANOMALIES AND DISEASES OF THE FIBROUS SYSTEM.

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SHALL confine my observations, in the following chapter, to the mem-
ous, and the fasciculated fibrous structures, except in the instance of
steum, which I shall consider as fully as can be done, without enter-
nto the subject of diseases of bone. The morbid conditions of the
ining fibrous structures will be duly noticed, according to their im-
nce, in connection with those organs and apparatus, of which they
either the capsules, the connecting medium, or the fundamental
ture.

1. *Deficiency and excess of development.*—Fibrous membranes are
letely wanting in all cases in which the organs or apparatus to which
belong do not exist: and, in like manner, a ligament or the tendon
muscle is sometimes absent altogether. But occasionally there is
tual deficiency of either kind of fibrous structure; it is merely back-
in its development, and is thin and weak, and resembles cellular
e.

hen double organs or apparatus exist, the fibrous structures are
le too; and accessory ligaments, additional tendons of muscles, &c.,
l other instances of plurality in this system. Moreover, there are
r circumstances under which fibrous structures are developed in un-
ral situations; of which we find instances in the new articular cap-
of false joints; in the thick dense plates, the tough bands, and the
rounded or branched, callous masses of fibroid tissue into which
acts of inflammation and coagulable lymph are converted, when they
ffused upon serous and synovial membranes, upon the internal coat
e vessels, or within parenchymatous tissues; in cicatrices generally;
e anomalous callus of bone; in the abnormal synchondroses formed
fractures; and lastly, in the fibroid tissue which composes the walls
sts and cystoid growths.

2. *Anomalies in size and form.*—Congenital anomalies of size are
nted both by fasciæ and ligaments, the former being unnaturally
acted, the latter too short: and in the instance of ligaments, it may
en that one or all of the ligaments of a joint are shorter than natu-
Sometimes the shortening of ligaments or tendons is brought on
birth by some loss of substance, or want of extending power, or by
nge in their texture.

At any period of fœtal, or of subsequent life, fibrous membranes may become enlarged; and this will occur, whatever be the cause of the distension or swelling of the cavities or organs they enclose; thus the sclerotica, the fibrous capsule of a joint, &c., may be found enlarged. Sometimes the membrane becomes proportionally thinner; at other times it increases in thickness and is hypertrophied, just as is the case, under similar circumstances, with the capsule and fibrous structure of certain parenchymatous organs, of which the chronic enlargements of the spleen are an example.

Moreover, the ligaments of joints are liable to relaxation if they be immoderately stretched for some time: and in paralyzed limbs they lose their tone, and elongate.

Fibrous membranes sometimes yield only in one direction; thus in hernia cerebri the dura mater expands only where there are apertures in the skull.

The anomalies of form are merely such as may be deduced from what has been said already, or such as consist of a few rare varieties in the shape of ligaments; any of which may be fissured or subdivided.

§ 3. *Anomalies of consistence and continuity.*—With the exception of a somewhat looser or closer texture of the ligaments, no alterations take place in the consistence of fibrous structures, which are not consequences of palpable disease of texture.

Amongst the solutions of continuity, those lacerations of ligaments and tendons are worthy of remark which are produced by external force and by excessive muscular action: they are more likely to happen if the tissue of these parts have been previously softened by inflammation; when indeed they may occur upon the slightest movements.

Fibrous membranes may be ruptured by excessive distension of the cavities which they enclose, or by immoderate congestion of a parenchyma contained within them, such for example as the spleen; and the same accident may result from their being struck or crushed. Concussion and contusion also sometimes lead to serious consequences when they separate periosteum, or the dura mater, from bone, as they give rise to hemorrhage into the interspace, and subsequently to inflammation and the effusion of a sanious product, as well as to necrosis.

Incised wounds of fibrous structures, especially of tendons, readily heal, as recent experience proves. An exudation from the wounded tendon and other injured parts fills the space which is left between the surfaces of the wound by the retraction of the muscle, and at first unites together all the neighboring structures that take part in the reactionary process; but afterwards it gradually becomes isolated, and new fibrous tissue is formed within it. Losses of substance are repaired in the same way, not only those which have been produced by external violence, but such also as have been caused by an ulcerative process. This fact may be observed in periosteum, or in the dura mater.

§ 4. *Deviations from natural texture.*

1. *Inflammation.*—Inflammation in fibrous structures is a frequent result of stretching and various kinds of injury, as well as of mere expo-

sure. Not less frequently it extends to them from other organs, such as bones and parenchymatous structures, in which inflammation or suppuration is taking place; and, as fibrous structures sometimes lie immediately beneath a serous membrane, they may become involved in an inflammation of it in the way described at p. 30. Lastly, fibrous structures may themselves inflame: in which case, several parts of the system are usually attacked, either together or in succession. Inflammations of this kind are attributed to rheumatism, and to many other constitutional maladies which are described as gout, syphilis, and so forth, but they still require much elucidation.

Inflammation in fibrous tissue is sometimes an acute disease, but very frequently its course is chronic. Its characters are as follow:

It begins with the appearance of streaks of injection, and here and there of red dots which consist of small quantities of extravasated blood: the diseased structure loses its peculiar lustre, and becomes tumid, being infiltrated with a fluid of a grayish or yellowish color, and partly jelly-like and coagulating. If the inflammation be violent, it gradually obliterates more and more, the longer it continues, all appearance of fibrous texture; the structure becomes easily lacerable, and the inflammatory product in it, most of which has coagulated, changes its color to a dirty yellowish-red, or reddish-brown. In this state it resembles, as has been remarked by Gendrin, an inflamed lymphatic gland,—i. e. chronic inflammation of a gland.

The neighboring tissues always share in the inflammation of a fibrous structure, but their relations to each other are altered in various ways. The homologous cellular tissue adjoining is usually inflamed in a considerable degree; and it becomes so confounded with the fibrous tissue, that the limits of either cease to be distinguishable. It is in this manner that inflamed fibrous structures—tendons and ligaments—are sometimes fixed in their bed of cellular tissue. But inflamed periosteum, on the other hand, and inflamed dura mater may be easily separated from the bone to which they belong; and some of the tunicæ albugineæ can be torn from their proper parenchyma, as if it were heterologous tissue. The loosening of the connection is proportioned to the acuteness and violence of the inflammation in the fibrous structure.

If inflammation attack any fibrous structure, such as the dura mater or the capsule of a joint, in consequence of its being laid bare and exposed to contact with the air, it first becomes reddened, dull and villous, and then granulations appear on its exposed surface, which unite with it into a uniform, red, flesh-like, soft mass. Sometimes, especially in tendons, this does not take place until a superficial layer has perished and been cast off. The granulations change into cicatrix tissue, and by it the fibrous structure unites with, and is fixed to, the cicatrix in the other injured organs, muscles, integuments, &c.

There are numerous terminations of inflammation in fibrous structures, and their occurrence is determined by various circumstances.

Suppuration ensues chiefly when the inflammation has been caused by exposure to the atmospheric air, and various other external irritants.

And, under similar circumstances, the inflammation leads to *ulceration*. Such is the result of inflammations which have been produced by the advance of some neighboring ulcerative process to the fibrous structure; as is instanced in ulcerations of periosteum or of the dura mater, when caries encroaches upon them; or of fibrous capsules, when suppuration is taking place in the serous and synovial membranes which adjoin them. The suppuration and ulceration, in such cases, mostly advance from the surface to the deeper parts of the fibrous tissue; while, at other times, collections of pus and sanies are found in its interior. An ulcer in fibrous structures sometimes has a sarcomatous or fungous appearance, in consequence of the flesh-like substance with which the tissue around it is infiltrated, and of the granulations which project from it.

Slight relapsing inflammations, and those which run a chronic course, end in *induration and thickening*. The soft, red, flesh-like tissue, infiltered with inflammatory product, becomes pale and contracted, and gradually changes to a white, dense, firm, fibroid mass, which looks like cartilage. This mass generally unites closely with any neighboring tissue, with bone, for instance, which has been inflamed at the same time, and can then be separated from it only with much difficulty; but the adhesion afterwards becomes much less strong, and may, indeed, at length be entirely destroyed, if the change now described in a fibrous membrane, and the contraction and obliteration of vessels to which it leads, produce attenuation of the tissue which the membrane encloses, or if, as sometimes happens, the inflammation of an organ enveloped in a tunica albuginea be followed by a secondary atrophy of its parenchyma.

Fibrous tissue, when indurated in the manner just described, not unfrequently becomes the seat of *ossification*: that is to say, of a calcareous deposit. It is observed especially in fascicular structures, such as the ligaments, but is very rarely met with in fibrous membranes, though we find it now and then in the dura mater.

Primary inflammation of a fibrous tissue very seldom terminates in *gangrene*; but it is a frequent occurrence when other neighboring structures, integuments, cellular tissue, muscles, &c., are sloughing too: it is so in the instance of bed-sloughs. The natural fibrous tissue changes into a blackish-brown mass, which is soaked through with sanies of the same character, is as soft as tinder, and may be torn in any direction. It degenerates in the same manner when it has been crushed or stretched, or when those tissues which convey its bloodvessels are torn off from it, and particularly when the removal of the cellular tissue exposes it to the external air.

Inflammation frequently takes place in periosteum. In its origin and character it corresponds with what has been said of inflammation of fibrous structures in general. It may coexist, even from its commencement, with inflammation of the bone, but it often originates as a substantive disease in the periosteum itself, and in that case always extends to the surface of the bone; or again, it may spread to the periosteum, either from the bone within, or from the soft parts without, especially from cellular tissue, ligaments, and fasciæ.

It is distinguished by the same characters as inflammation of other fibrous structures. The membrane unites closely with the cellular tissue which takes part in the process, and thus with the adjoining structures, with sheaths of muscles, aponeuroses, integuments, &c.: but from the bone it can be stripped with ease, especially if the inflammation have been at all intense, or have extended along the prolongations of the membrane into the bone, and have led to any exudation on its inner surface. Sometimes, indeed, the periosteum is found separated from the bone by a considerable quantity of purulent or sanious exudation.

Under whichever of the above-named conditions inflammation of periosteum occur, it frequently proceeds to suppuration and ulceration. There are various ways in which these results may ensue: sometimes they commence at the outer surface of the membrane, sometimes in its interior, sometimes between it and the bone; sometimes again they take place in circumscribed spots, which gradually enlarge and coalesce, while at other times extensive tracts of the membrane are found rapidly undergoing solution. (Schmelzung).

Periosteum is easily replaced when it has been lost by injury; and even when it has been destroyed by ulceration, so soon as the constitutional cause of the inflammation of the bone has ceased. The new membrane is formed out of a plastic exudation from the bone.

Chronic inflammation often leaves behind it thickening and induration of the periosteum. That membrane is then found changed into a whitish layer, which may be several lines in thickness: its texture is very close, and it is as tough as leather, or fibro-cartilage. It adheres closely to the bone, and seems intimately united with it. The condition of the bone under such circumstances varies considerably, and will be described in another chapter.

2. *Adventitious growths* are, on the whole, but rarely found in this system, though there are some fibrous structures which form an exception to the rule in the instance of particular new growths. Such is certainly the case with the dura mater in respect to sarcomatous and cancerous growths. And it must also be remembered, that in all cases in which fibrous membranes are implicated, it is extremely difficult to determine whether the new, and the degenerated growths which are imbedded in their tissue, were originally formed in them, or were developed, as undoubtedly may be proved to be far more frequently the fact, in the parenchymatous organs which those membranes enclose.

a. Cysts.

α. Single cysts of small size are not uncommon in the structures belonging to the fibrous system. Their contents may be serous, or like synovia, viscid, or gum-like, or they may consist of cholesterine. Cysts with serous contents occur chiefly in tendons in and between aponeurotic expansions and fasciæ, and in periosteum: those which contain cholesterine are most common in periosteum, where they are known by the name of encysted cholesteatoma.

β. Compound cystoid growths are very rare: I am acquainted with but one such case. It is that of an old and ill-preserved specimen of cystoid growth, some of which adhere to the periosteum, while others are contained in the muscles of the same lower extremity.

b. Fibroid tissue is formed in fibrous structures, especially in ligaments and periosteum, when they have been swollen and thickened by chronic inflammation: it is also found in what are called fibrous tumors. These tumors are met with chiefly in periosteum, and in the dura mater: and they present the various characters of the soft, succulent, and delicately fibrillated tumor, filled with numerous elementary cells; of the spongy tumor composed of interwoven celled fibres, and of that which is dense and compact, and consists of well-formed fibrous bands. Their size is very uncertain, varying from that of a pea to that of a walnut or a fist; in a few cases, especially of genuine compact fibroid tumors, they are even as large as the head of a child.

c. Fibrous structures *ossify* in various ways. Not only are they liable to calcareous deposition, but when hypertrophied and indurated after inflammation, they sometimes have osteoid tissue developed in them. Sometimes it is formed in the substance of the fibrous structure, and resembles needles, cords, or plates, or is altogether shapeless; at other times it is a more or less complete incrustation upon its surface. Under such circumstances, a certain amount of vascularity is observed in the fibrous structure; it becomes of a brownish-red color, and remains, after the bony matter has been deposited, blackened with pigment, and uncommonly dry. Bony growths of this kind are formed for the most part in the fibrous capsule of the spleen, in the dura mater, and in the fasciculated articular ligaments. Of course similar productions are found in the fibroid tumors of fibrous organs.

But not only is osteoid tissue produced in fibrous organs; in some cases we find true bone in them. The exudations from periosteum and from the dura mater on the surface next the bone, become, when they ossify, normal bony tissue. They form broad, thin layers, or thicker, circumscribed plates and shapeless masses, and enter, for the most part at once, into organic connection with the bone beneath them. They are all included under the name of osteophyte and exostosis, whether occurring as a growth of bone in various tendons, as ossified callus in articular capsules after the occurrence of fractures within joints, or as ossification of fasciculated ligaments in cases of anchylosis, &c.

d. Tubercle.—Tubercle is on the whole but rarely met with in fibrous organs: and when it does occur, it is almost only on the periosteum of spongy bones, and on the dura mater. In the usual mode of its origin, inflammation gives rise to a tubercular product, which is deposited in the tissue of the membrane, or on that surface of it which adjoins the bone; it degenerates into a mass of caseo-purulent matter, and being enclosed in a capsule of fibrous structure, which is infiltrated with lardaceo-gelatinous substance, it forms loose, pulpy swellings. By the progressive formation and degeneration of tubercles in the adjoining fibrous tissue it advances in the membrane, and to the bone; and frequently produces, especially in the spine, destructive ulceration of the periosteum and ligamentous apparatus along an extensive sinuous track, as well as caries of the bones.

Occasionally these sinuses close, and their contents become chalky. They are very commonly associated with tubercle in the cellular tissue, and in the lymphatic glands, and very often with tubercle in the lungs. This subject must be resumed in the chapter on the Diseases of Bone.

e. Sarcoma and cancer.—All fibrous structures may be destroyed by adjoining cancerous disease. In periosteum and the dura mater it occurs also as a primary disease, and from the former structure especially, it soon advances to the bone. Fibrous and medullary cancer are the kinds mostly observed in periosteum, but those which occur in the dura mater are very various, and differ remarkably in their elementary structure. Further remarks on this subject will be made in the chapters on the Diseases of the Bones, the Joints, and the Dura Mater.

Note.—Some diseases have been described under the names of gummata, periostoses, and Cooper's cartilaginous and fungous exostoses; they will be found to be either a circumscribed inflammatory swelling and induration of the periosteum, or one of the adventitious growths which have been already described as occurring in it, or they will correspond with some of those to be hereafter brought forward amongst the diseases of Bone.

PART IX.

ANOMALIES AND DISEASES OF THE OSSEOUS SYSTEM.

PART IX.

ANOMALIES AND DISEASES OF THE OSSEOUS SYSTEM.

CHAPTER I.

ANOMALIES AND DISEASES OF BONE IN GENERAL.

§ 1. *Deficiency and excess of development.*—The entire bony fabric of the body has been found wanting in some few cases of monstrosity, and even in some individuals whose development in other respects was quite natural. A partial deficiency of the skeleton is less unfrequently observed, as it occurs in various parts of the body where development generally has been arrested; in the thorax, for instance, and the pelvis, and especially in the limbs. There is very commonly no bone in supernumerary peripheral parts, whether fingers, toes, or limbs.

Moreover, the skeleton very often falls short of its complete development, in being altogether cartilaginous, or at least very imperfectly ossified, at the time of birth. This condition, known as congenital rickets, sometimes continues to a later period of life, and betrays itself by insufficient firmness and power of resistance in the bones, by persistence of those characters which belong to the skeleton in childhood, by the apophyses remaining separate, &c. It is very commonly combined with hypertrophy of the white substance of the brain. In some cases the complete formation of the bones is retarded by long-continued and exhausting diseases, and in some parts of the skeleton it is very frequently arrested, to a great extent, by pressure from within; as, for instance, in the skull.

An excess of development is exhibited, on the one hand, when the whole skeleton, or parts of it, are completely formed at an unnaturally early period; as when the fontanelles close, the sutures disappear, the epiphyses unite with the diaphyses, the teeth are cut, &c., prematurely; and, on the other hand, it is seen, also, when the bones are unnaturally dense and hard, when they grow out and enlarge in some unusual direction, or when various new bony formations are found upon them.

The bones may vary in number either way; they may be more numerous or fewer than natural. When certain parts are wanting, or exceed their natural number, the corresponding bones are wanting or supernumerary too: but this may be the case also when a part appears to be naturally formed, as we find exemplified in the toes and sesamoid bones, in the vertebræ, and the ribs. The most frequent instance in which the number of the bones is usually great (although it is only an apparent excess), is when the pieces of which a bone is composed continue separate, or when, in the skull more particularly, unusual sutures, or sutural (Wormian) bones exist. The want of a bone is sometimes made up for by a supplementary increase in the bulk of a neighboring bone.

§ 2. *Anomalies in size.—Hypertrophy and Atrophy in particular.*—The anomalies of this class present many varieties, both when the volume of the bone is greater than natural and when it is less.

An *increase* in the size of the bones occurs in various forms.

1. They may grow to a greater length than natural. In giants this excess prevails in the whole skeleton; but sometimes it is observed only in particular fingers, toes, or limbs; in the latter case, it may be congenital, or may come on after birth, during the period of childhood or youth.

2. The increase may take place in the breadth and thickness of a bone, at the same time that its texture becomes more dense, and its weight greater. This is hypertrophy, hyperostosis; it may extend over the whole of a bone, or be confined to particular parts of it.

3. The enlargement of a bone may be a consequence of expansion of its texture—of dilatation of its Haversian canals, and, in long bones, of the medullary cavities. It is by this change that the enlargement of cavities which are made up of bones, especially of the skull, is effected: it arises from pressure on the bones from within.

4. Enlargement is sometimes occasioned by the development of various kinds of adventitious growths and tumors in bone.

The volume of bones is *less* than natural,—

1. When the whole skeleton or a part of it, as the head, or one or more of the limbs, has failed in attaining its full growth, whether its small size be observable at birth, or be first exhibited at a later period in a dwarfish growth of the body. Congenital dwarfishness is distinguished generally by an arrested development of the extremities in length, which is at once seen on comparing them with the skull and vertebral column; by the striking appearance of thickness which the articular ends of the bones consequently obtain; and by their deformity generally. Although congenital dwarfishness manifests on the whole a seeming alliance with those changes which are produced by rickets in the growth and form of bones, yet it cannot be certainly said to be due to the existence of that disease in the fœtus: its pathology is still unsettled.

2. Or else the small size of bones proceeds from atrophy.

A. *Hypertrophy—Hyperostosis.*—Bone increases in substance in two ways, which are not essentially different from one another.

1. In the one case, while the density of the bone remains unchanged, new osseous substance is deposited on its surface, beneath the periosteum, and augments it in breadth and thickness. The size of the medullary canal remains the same, but the compact substance around it is thicker than before (External hyperostosis).

2. In the other case, the increase of substance is internal, proceeding from the Haversian canals, and in the end from the whole medullary system. The bone becomes more dense, not only in its compact layers, but also in its cancellous part; the walls of its cells, and the bony

threads of which its network is composed, increase in thickness; and, by a kind of concentric hypertrophy, as it were, the medullary cavity diminishes in size, and the diploe disappears. We may call this state an internal hyperostosis; it constitutes also the induration (sclerosis) of bony tissue.

The two forms very commonly occur together, and thus in a twofold manner augment both the bulk and the weight of the bone.

Each is the result of the gradual formation of too great a quantity of the cartilage of bone, in which the normal salts of lime become deposited: both affect compact bones, as the cranial bones, the central piece of the long bones, chiefly but not exclusively; for a somewhat striking density and resistance of the spongy bones also, especially of the vertebra, is frequently observed in persons about the period of youth and manhood. They become very important diseases, when they proceed to any great extent, or involve important parts of the skeleton, such, for instance, as the skull; or when they affect large portions, or the whole, of the skeleton. No previous disease has occurred in the texture of the bone, which can be regarded as a preliminary or a causative process: its surface is level and smooth, the periosteum is natural, and even when the induration attains the density and hardness of ivory, the bony texture is, in other respects, natural. Nevertheless, it must be remarked, that in advanced cases of hyperostosis, the new ossifying substance is very commonly deposited unevenly, more at one part than at another, and that thus, at length, the bone acquires a misshapen and coarse appearance.

It is sometimes observed, that while one bone is in a state of hyperostosis, another is wasted: this is frequently the case in the skull, where, when the cranial vault is hypertrophied, the bones of the face and of the base of the skull are atrophied.

In other cases, the increase of substance, both the internal and the external, is occasioned by an inflammatory process. Sometimes the inflammation attacks the outermost layer of a bone and the periosteum; sometimes it affects the deeper seated capillaries, and sometimes the medullary membrane. In the first cases it produces an exudation on the free surface of the bone, which becomes converted into an osseous layer, compact like the surface on which it is effused, though sometimes separated from it by diploetic spongy substance. In the last two cases, the increase of substance is within the bone, and leads to induration, either of the compact tissue, or of the inner spongy portion. These conditions have been called by Lobstein, sclerosis supra-corticalis, corticalis, and centralis respectively; they are, however, very rarely independent and separate, though one form frequently predominates in one or other portion of the bone. Generally, before they take place, the texture of the bone becomes expanded in consequence of swelling and infiltration of the tissue which connects the capillaries of the medullary canals and cells —that is, those spaces are dilated: and as the process usually occupies only single bones, and is not uniform in its degree at all points, the hyperostoses which result are also confined to single bones, the diseased bone is thicker than natural, and very often is, from the first, of unequal thickness and coarse: its external surface is uneven and rough, nodulated and full of fissures, like the bark of a tree, or covered with thin, leaf-like inequalities; it is rendered porous by the permanently dilated orifices for its vessels, &c.;* and the periosteum, and its prolongations inward, are thickened and hypertrophied.

The hyperostosis and indurations belonging to this class, are for the most part results of chronic inflammations, especially those of a syphilitic or gouty character. I shall have some general remarks to make upon them in a more suitable place, when considering the question, whether the hyperostoses, which these diseases set up, bear any marks by which they can be distinguished from one another.

Lastly, there are yet other cases in which hyperostosis, especially the internal (the induration), is occasioned by previous softening and expansion of the texture of the bone. The secondary indurations will be considered with the diseases of the texture of bone, that they may be arranged amongst, and shown to arise from, them; for those indurations have their origin, not merely in the ordinary ossification, within the bone, of a too abundant cartilage, but most probably, either in the ossification of a diseased cartilage, whilst the earths and salts which are deposited, are abnormal as well as normal, or in overloading the cartilaginous basement with salts, which yet are normal. In either case the anomaly is one of texture.

Hyperostosis presents itself further in different forms as a local disease, confined to small spots on one or more bones. Its two principal forms, though intermixed in some respects, by their transitions into one another, are yet, in many essential points, distinct: they are the common exostosis or bony excrescence, and the new growth, which has been named by Lobstein, the osteophyte.

a. Exostosis.—By the term exostosis should be understood a purely bony mass, set upon a bone forming with it an organic whole, and, where it is possible, originating, or proceeding, from the bone. When its development is complete, and often at the beginning of its growth, its texture is always homologous with that of its base and point of origin, whether that be compact or spongy. Hence all new growths upon or within a bone, which hold any other relation to it, are excluded, although they be composed more or less of normal bony texture, and even although composed of such texture altogether. Bony growths, however, which proceed from the periosteum, but sooner or later become united with the bone, are admitted.

The most important varieties in an exostosis relate to its texture, to the point from which it originated (though in many cases this cannot be determined with certainty), and to the mode of its development. To these varieties the sundry and wide differences in its size, form, mode of attachment, &c., are mainly referable.

Exostoses are composed sometimes of compact, sometimes of spongy bony substance; and although some are made up of both these substances, yet the division into those which are compact and those which are spongy is so far valuable, that it expresses their original condition and their development.

α. The most frequent of the exostoses is the compact. It occurs on compact bones and parts of bones, particularly on the outer table of the cranial bones.

It appears as if it had been planted on the surface of the bone from without: in general it is a plane convex nodule, the margin of which is abrupt, and often separated from the bone beneath by a furrow. This furrow is generally narrow, sometimes being but just perceptible, and about the thickness of a hair; but frequently it is deep, and forms a fissure between the tumor and its basement. It gives the exostosis the appearance of having been glued on, or of sitting, mushroom-like, on a very short stalk.

Not only is this exostosis in all cases compact, but it often exceeds in density the bone from which it springs: it is then known as the ivory exostosis. It is especially liable to be formed on bones which are themselves indurated.

It is compact from the very first; and grows in such a way that the layers which are added to it always at once become as dense as ivory. Neither the most superficial and most recent strata, nor the smallest of those exostoses, which form near larger ones, even though no larger than hemp-seed or a lentil, is ever seen to contain any spongy structure. New layers and old, large exostoses and small, are equally dense and hard. When they are minutely examined, the number of peripheral lamellæ is found to be very considerable; and the corpuscles lying amongst them are long. The Haversian canals are small and far apart, and many of them are surrounded by a distinct and completely defined (völlig abgeschlossenen) lamellar system. With regard to the corpuscles, we find large tracts without any of them, while at other spots they are clustered together in dense groups.

The number of these exostoses occurring in one person, and even on the same bone, is sometimes very considerable; especially if the very small ones, which are easily overlooked, be also enumerated. I have met with them almost exclusively on the skull, where, like induration, they do, in fact, most frequently occur; but they are likewise observed on the long bones, and on the bones of the pelvis.

They vary in size from that of a flattened hemp-seed or a lentil, which is scarcely perceptible, to that of a walnut, or a hen's egg, and even to greater dimensions. Their most common size ranges between that of a pea and that of a hazel-nut.

While their usual form is that of a plane convex nodule, their surface, whether even or uneven, is always smooth and polished. If they grow beyond the ordinary size, they become round, or oval, or, as they generally rather increase in length, they form a more or less cylindrical, horn-like projection. There is another form which occurs with them on the inner table of the skull near the frontal crest: it has a peculiar humifuse[1] character, or the appearance of a convoluted wreath.

The color of these exostoses is white or yellowish-white,—whiter than that of the bone to which they are attached.

As we cannot associate exostoses, in respect to their cause, with the various inequalities and nodules that occur on bones from constitutional disease, especially from syphilis, the occasion of their origin must be

[1] ["Humifuse"—growing parallel to the surface, but attached only at its point of origin, like the stems of plants which creep along the ground without taking root. (Palmer's Pent. Dict.)—Ed.]

said to be unknown. As I have already remarked, they are very generally found on the bones of the skull, of which one or more are at the same time the seat of induration, and not unfrequently bony formations are found also on the dura mater.

β. The spongy exostosis proceeds from a circumscribed rarefaction, or expansion, of the bony tissue (osteoporosis); it forms a tumor of cellular texture abounding with marrow, which is surrounded by a compact layer or rind. It is sometimes developed from compact bony tissue, sometimes from spongy substance, and either from the peripheral laminæ of the bone, or from its interior. It presents, accordingly, many striking varieties of external form and of internal structure. Its rind or external layer unites with that of the bone; its surface is uneven.

Sometimes it forms a slight, rounded elevation, above the surface of the bone, sometimes a more sharply circumscribed, hemispherical tumor; or it has a still narrower base, and is globular.

We not unfrequently find, near or upon the articular ends of long bones, and especially on the tibia and femur towards the knee-joint, a rounded, gnarled, and uneven excrescence, sometimes lobulated, or branched, and set upon a well-formed stalk; sometimes it has the form of rounded or angular, thorn-like processes. Such excrescences may be occasionally found near the articulations on most, if not all, the long bones of a skeleton.

Lastly, there is another form of exostosis allied to the spongy form, which has not only a spongy texture within its compact rind, but also a well-formed medullary cavity communicating with the medullary tube and cells of the bone; and thus presenting, as it were, a dilatation of the medullary cavity beyond the surface of the bone. Whether such really be the mode of origin of this form of exostosis, and a central cavity of this kind, communicating with the medullary tube of the bone, really exist from the commencement of the disease (in which case this exostosis would clearly rank with the spongy form), or whether the central cavity be formed in it subsequently, as it is in callus, is as yet unknown.

In the skull, the expansion of the diploetic structure sometimes distends both the compact tables, and thus there is an internal exostosis corresponding with the external.

The spongy exostosis continues for an indefinite period in its original spongy state: not unfrequently perhaps it may remain so permanently; but more commonly new substance is deposited in its interior, and more or less induration ensues. The compact exterior wall acquires considerable thickness, and encloses a mass of cancellous substance, or a well-formed central cavity: sometimes spots in its interior are found compact too, or it may even become uniformly solid throughout.

In some parts, and even in the whole of the exostosis, a renewal of rarefaction, or osteoporosis, very often appears to succeed this process of condensation. It may be in this manner that the growth of the spongy exostosis from within outwards is chiefly effected.

They very often grow to a considerable size.

Exostoses are formed, as a general rule, in the outer layers of a bone, and they grow and project outwards: but, in a few cases, they have been seen to advance in the opposite direction, and protrude within the

can be determined by the characters which the osteophyte presents. I shall have an opportunity hereafter of introducing what I have observed on this subject; it may, perhaps, be deemed a contribution to our knowledge of diseases of bone, in its present incomplete state.

In the first place, Lobstein has conceived that a distinction should be drawn between exostosis and what he denominates osteophyte. I shall not, however, follow his subdivision, but shall attempt to give a more practical representation of the disease.

a. The *velvety villous* osteophyte sometimes appears as a single and very thin layer, resembling a coating of hoar-frost; sometimes it measures one or two lines or more in thickness. It seems to be composed of delicate fibrils and lamellæ, which are fixed at acute angles on the surface of the bone, and give it the appearance of velvet, or of felt with a very fine nap. As it increases in density it gradually acquires a smooth exterior, which is pierced with very numerous fine pores; while its deeper structure is more distinctly laminated. Although, at first, in contact with the bone, it is usually almost entirely unattached to it, and can easily be raised in large pieces from its surface. The osseous surface under it is sometimes as smooth as in the natural state; but sometimes we find, upon close examination, that it has lost its smoothness, and is distinctly rough, the pores for its vessels being somewhat enlarged, and its outermost layer here and there expanded into filaments. At a later period, the osteophyte is found attached to the bone by some intervening minute round pillars and plates: after having gradually become compact, it unites with the bone. A layer of cellular (diploetic) substance forms for some time a line of demarcation between it and the bone, but when this disappears, the osteophyte and the compact surface of the bone compose one uniform whole. It corresponds to the "osteophyte diffuse et fibrillo-reticulaire" of Lobstein. Sometimes, as an exception, it is produced in more considerable quantity, and forms an intimate connection with the bone, uniting with the filamento-cellular structure into which the outer layer of the bone expands.

Should the periosteum have taken part in the process by which the osteophyte is produced, that portion of the exudation which attaches to it, becomes organized into a vascular cellular tissue, while that which belongs to the bone, and which ossifies, forms a compact osseous layer: the surface of the latter presents a great number of pores, and of exceedingly tortuous and convoluted half canals, which have been occupied by the newly-formed cellular tissue, and its vascular twigs. We notice this chiefly when such an osteophyte is situated on the inner table of the skull.

The color of this osteophyte in the recent state depends on the intensity of the process which produced it, on the period of its existence, and on the progress of its ossification: and it varies accordingly from bluish or rosy-red to yellowish and dirty white, or it may be a dazzling white, and glisten like silk or asbestos. At its commencement it is a soft, gelatinous exudation; afterwards it becomes tough and elastic, like cartilage; and finally it ossifies.

This kind of osteophyte is seen chiefly on compact bones. It accompanies almost all inflammatory processes, abscesses, and necrosis in bone, especially in young persons with abundant fluids (succulent): it frequently extends from the diseased spot over considerable portions of the surface of the bone, while it accumulates in greatest quantity in the immediate neighborhood of the disease, and occasionally on the rough lines, &c. It is very frequent in the skull, especially on its inner table; and on that part particularly there is a bony growth, which has some connection with pregnancy, and which we call the *puerperal osteophyte.* It possesses great interest, and will receive a fuller consideration when we come to treat of the diseases of the cranial bones.

β. Allied to this osteophyte is another which I will call the *splintered and laminated osteophyte.* It presents itself in the form of rather large excrescences and lamellæ, which measure several lines in length, are conical in shape, and mostly terminate in a sharp point: they are composed of a compact wall, pierced with fine pores, which encloses a coarsely cellular, osseous tissue, or even a single (medullary) cavity. It is chiefly, and indeed commonly, abundant in the neighborhood of the cancellous parts of a bone affected with caries, especially caries of the articular ends of bones in young subjects, which is generally considered to be of scrofulous nature.

γ. *Warty and stalactitic osteophyte.*—This is a bony growth either of excrescences resembling warts and attached sometimes by a broad base, sometimes by a pedicle, or of larger, irregular, rugged masses resembling stalactites. It seems to be found only in the neighborhood of the joints; it accumulates particularly around enlarged articular concavities, forms a tuberculated addition to the margin of articular heads, which have been flattened out like a mushroom, and thence extends in abundance also to adjoining rough parts of the bone. They generally consist of a chalky, white, very brittle substance; they occur most commonly on the hip bones, and appear to be connected with gouty metamorphosis of that joint.

δ. The osteophyte in the form of thorny or styloid, single or branched plates, or of rounded, gnarled, and pediculated processes, occurs chiefly on spongy bones, on the vertebræ, and the bones of the pelvis, selecting in both these instances the neighborhood of the synchondroses. It occurs also on the articular ends of bones, and very frequently follows, as it grows, the direction of the fibres of the periosteum, ligaments, tendinous insertions of the muscles, intermuscular septa, interosseous membranes, &c. When growths of this kind spring from two adjoining bones, they frequently meet somewhat in a symmetrical manner, and unite together: in this manner they enclose the synchondrosal cartilage in a more or less complete bony capsule, and give rise to a peculiar kind of anchylosis, which is often observed on the bodies of the vertebræ. Not unfrequently they supply the place of callus which has been arrested in its development, and imperfectly unite the ends of broken bones. Similar formations are sometimes seen around the cloacal openings in the capsule of a sequestrum. They arise from a chronic inflammation of the bones, in which the periosteum and ligamentous apparatus become involved: they are often found on the vertebræ of old persons, and while the bone to which they are attached, as well as some others, or even the whole skeleton, is atrophied, the osteophyte itself is of dense structure, and hard.

ε. There is another osteophyte, which looks as if a quantity of bony matter had been poured over a bone, and had coagulated as it flowed. It forms masses which sometimes give the impression, that the bony substance had been *dropped* upon a bone, and had then solidified; in which case the surface of the mass, whether even or uneven, is smooth: at other times it appears as if the bony matter had been *poured* in a stream over larger surfaces of a bone, and had then coagulated. This osteophyte is compact. I have seen it on the inner table of the skull, when the cranial bones were indurated; and have met with it still more frequently covering a considerable extent of the anterior surface of the vertebral column in old persons, and producing anchylosis of the vertebræ.

In accordance with the foregoing descriptions, I exclude from amongst the osteophytes all those bony growths which form a more or less complete external capsule, or an internal radiated thorn-like skeleton, for the various adventitious structures that occur in bone, whether enchondroma, osteosarcoma, or cancer; as well as those which are found at the base of similar adventitious structures in the softer organs.

Osteophytes have in some few cases been observed covering large portions, and even almost the whole of a skeleton. Sometimes they are accompanied by hypertrophy, sometimes by atrophy of the bones. We are quite ignorant of any general diseased condition of the system to which this can be attributed.

The periosteum may not share in the diseased process going on in the exterior of the bone, and then it remains nearly unaltered; but if it do take part, it becomes vascular, reddened, infiltrated, and thickened (hypertrophied), and furnishes sheath-like prolongations, which invest the more bulky of the bony excrescences.

The changes which the osteophyte undergoes in the course of time have been already partly noticed. The fibrils and lamellæ which compose the velvety osteophyte approach each other and increase in size, and, at the same time, assume a position parallel to the lamellæ of the bone beneath them; the whole osteophyte becomes more dense, and unites either immediately or by an intervening layer of diploe, with the bone. Hence the bone becomes a layer of compact tissue thicker—Lobstein's "supracorticale osteosclerose." In other cases, as the velvety, or the splintered and laminated, osteophyte becomes condensed, its fibrils and lamellæ retain their relative position to the bone, and the osteophyte becomes a compact osseous mass, which, though attached to the bone, can be distinguished from it by the different direction of its lamellæ and the course of its medullary canals. Sometimes the osteophyte appears to be diminished by absorption, but it can hardly disappear altogether.

B. *Atrophy.*—Atrophy of bones occurs under many forms and various circumstances.

(1.) After a long-continued and exhausting disease of bone, such as caries, after exhaustive healing processes, such as fractures (and injuries generally), or in consequence of palsies, neuralgia, or anchylosis, single bones, or large portions of the skeleton may diminish in volume. They retain their normal texture, but diminish in length, and still more in thickness; an entire bone becomes small, and its medullary canal contracted,—it is in a state of *concentric atrophy*. And in connection with this fact, and in opposition to that increase in the volume of bones to which I have before adverted as a consequence of distension of the cavity which they form, I may mention that bony cavities diminish, or sink in, when atrophy or loss of substance happens to the organs contained within them.

(2.) Bones are subject in old age to a form of atrophy (senile atrophy), in which their consistence and strength are so far changed that they not only become soft and flexible, and are easily indented, but rather acquire something of the brittleness of glass. (Fragilitas Vitrea. Osteopsathyrosis of Lobstein.)

Atrophy appears always to commence with the medullary tubes and diploetic structure: the cells of the latter enlarge, and its walls, as well as the bony threads composing the cancellous tissue, become attenuated, and at length disappear entirely. The compact substance yielding next, is all changed into spongy, diploetic tissue, except its outermost layer, but though that remains compact, it becomes extremely thin, and is sometimes scarcely as thick as a sheet of paper. As the atrophy of this once solid, but now spongy, substance becomes more complete, the outermost layer alone remains, encompassing either a cavity, which contains some mere traces of spongy tissue at its periphery,—a cavity relatively dilated (*excentric atrophy*), or a soft substance, with very coarse cells; or lastly, when the diploe is entirely removed, the extremely thin remaining parts of the walls of the bone approach each other, and coalescing form a single thin plate. Examples of the first change are presented by the larger medullary tubes; the second is seen in the smaller cylindrical bones, in the pelvic bones, ribs, and vertebræ; while the bones of the face, and small spots, which are limited to the top of the parietal bones of aged persos, exhibit the last. Finally, should the atrophy proceed to an extreme degree, and involve the last remaining thin layer of the wall of the bone, its surface becomes rough and porous; and, however easily the periosteum may be stripped off elsewhere, it cannot be removed from this spot without bringing away a layer of bone with it.

The medullary tubes and dilated cellular interspaces are filled with marrow, which is usually of a dark, and often of a brownish-red, or a chocolate color.

The description given above sufficiently explains how it is that the bones become unusually flexible, and easily crack when they are bent; that upon making moderate pressure on spongy bones the finger breaks into them; that the cylindrical shafts, being reduced to a thin compact wall, break upon the slightest occasions; and that, in advanced age, the well-known curvatures of the vertebral column occur, and other portions of the bony fabric of the body become, under certain circumstances, crooked and deformed.

As the bones lose substance in their interior, they shrink in their external bulk: and hence the skeleton in old age becomes smaller in all its dimensions, and the weight of the body less.

Senile atrophy runs a chronic course, and is unattended with pain; in its earlier stages the muscles also waste and the lungs become atrophied; and afterwards the diminution of the bones is attended with decay (involution) of all the other organs: the muscles are sometimes the seat of fatty degeneration. There are some diseases of bone, which, in their anatomical characters, present considerable resemblance to this form of atrophy; but, although hitherto thought much of, those characters are in themselves unsatisfactory. They are diseases which affect persons unlike those in whom senile atrophy is met with, and in their symptoms and course they materially differ from it. As they are processes connected with constitutional disease (dyscrasia), and we partly recognize in them, and partly have reason to assume, the existence of important qualitative deviations from a healthy state of the organization, I shall speak further of them under the head of Morbid Expansion and Softening of Bones.

(3.) A third form of atrophy is that in which bone is worn away or absorbed (Usura, detritus ossis). Being occasioned either by uniform and permanent, or by repeatedly renewed (pulsatile), pressure upon the bone, the breach of substance is always circumscribed. Various tumors, which form in the soft parts adjoining a bone, especially in periosteum, produce this effect with different degrees of force: the walls of the skull, for instance, are pressed upon by the Pacchionian bodies, by sundry adventitious growths, commonly known by the collective name of fungus of the dura mater, by tumors in the brain, even by the brain itself, when enlarged or displaced, by large apoplectic cysts, by an enlarged and diseased pituitary gland, &c.; the bones of the face suffer from the pressure of fibrous tumors (fibrous polypi), of sarcoma, and cancerous growths developed in the nostrils, frontal sinuses, antra Highmoriana, or orbits; but the most frequent cause of pressure is aneurismal tumors, and the common seat of it is the bones of the trunk and limbs.

The degree and the extent to which the bone is worn away varies in different cases, the former depending on the amount and duration of the pressure, the latter on the size of the tumor: one or more bones may be entirely destroyed, or a bony wall may be perforated, as is often strikingly illustrated in the progress of aneurismal tumors. As large tumors press unequally, the destruction of bone, when very extensive, is generally not uniform: for the same reason its boundaries are not sharply defined.

Small tumors, which exert uniform and very moderate pressure, and even larger tumors, when they grow slowly, occasion, first, flattening, and then an excavation of the bone on which they press, but do not disturb the smoothness and polish of its surface. The bone immediately around bulges out, and appears not as if it had sustained an actual loss of substance, but rather as if its substance had been merely thrust aside. When pressure is made on one of two compact tables, especially in the skull, it is not so much that table which seems to be absorbed, as the layer of diploe beneath it; the two tables are in this manner gradually brought nearer together, and at length come into contact and unite. I find this borne out by many well-marked cases in the University Museum at Vienna; as well as by most of the pits on the inner surface of the skull, in which Pacchionian bodies have been imbedded.

If the compact wall of a bone be subjected to considerable pressure, it disappears layer by layer, becomes rough on the surface, and when at length entirely absorbed, leaves the cancellous tissue beneath it exposed. A very manifest effort of nature is then often perceived to resist the injury, and to maintain the integrity of the inner texture of the bone.

The cancellous substance, increasing in density by the addition of bony matter to its lamellæ and threads, strives to become compact, and exposes to the pressure a stratum as capable of resistance as possible.

This wearing down (detritus) of bone may easily be confounded with the loss of substance which results from caries; and the difficulty of distinguishing between them is sometimes augmented by their both occurring together. For, to take a frequent instance, that of softening malignant tumors, not only is the bone worn away by the pressure which such tumors exert, but inflammation and suppuration are set up in its exposed spongy texture, or corrosion on its surface, by the ichor which they discharge.

Absorption (detritus) is distinguished from caries by the absence of any change of texture, either at the spot itself, or around it; and by there being neither purulent nor sanious product, nor any osteophyte. The tendency to condensation which is exhibited in the substance exposed to pressure furnishes a further distinctive mark of the detritus.

Finally, bone may be absorbed in consequence of pressure from within: the various tumors developed in the spongy substance, and medullary cavity, the fibroid tumors, enchondroma, osteoid growths, sarcoma, cancer, and the dilatations of the capillary system of the vessels, known by the name of teleangiectases or erectile tumors, all commence their ravages within the bone.

§ 3. *Anomalies of form.*—The deviations from the natural shape of the bones are many and various; in some instances they are congenital, very frequently they take place after birth. When congenital, they sometimes occur independently and alone, sometimes an apparatus with which the altered bone stands in intimate relation is malformed too; at other times the change of form, whether congenital or acquired, results from some disease in the bone itself, or in other structures. To avoid repetition, I do not enumerate them here; for the most important of them will be more conveniently described amongst the anomalies of the several parts of the skeleton, and of the bones composing them, as well as in the chapter on the Joints.

§ 4. *Anomalies in the relative position of bones, and in their connection with one another.*—The connection between bones is sometimes unnaturally close and intimate, and sometimes unnaturally loose: when the latter condition is very decided, it is usually combined with some deviation from their relative position, that is, with dislocation.

The former state, or that in which bones are bound too closely together, is found, both when their articulation with each other is movable, and when it is immovable. It is known as *synostosis* or *anchylosis*, though the latter term is chiefly employed to designate the fixed state of a joint. Synostosis is sometimes congenital, but much more frequently it is acquired.

Congenital synostosis may be the result of an unnatural fusion of points of ossification belonging to separate bones; it is then almost always manifestly prejudicial to the full development of one or both of the united bones, and it accompanies other and more important malformations, such as acephalus, cyclopia, &c.: or it may consist of premature union of bones, which do not naturally unite till various periods after birth: thus the cranial bones are sometimes found united even in the fœtus.

Allied to this, is the case in which certain bones coalesce at some period subsequent to birth, but earlier than that at which their union normally takes place. Thus the cranial bones sometimes unite prematurely with each other, and so do the two halves of the lower jaw, epiphyses with their diaphyses, &c.

Synostosis, when acquired, is either incomplete, that is to say, adjoining bones become bound together by bridges of new bone (osteophytes), which pass over the intervening synchondroses and articular cavities, and enclose them in a more or less perfect bony capsule: or it is complete; the synchondrosal cartilages, or the soft tissues of a joint, having been removed by atrophy, suppuration, &c., the bones are brought into immediate contact with one another, and become conjoined. Vertebræ, the pubic bones, or the bones composing a joint, unite thus with one another; so too does the sacrum with the ossa innominata.

Other bones, also, when brought into permanent and close mutual contact, may become fixed together in the same manner; the ribs, for instance, in cases of lateral curvature.

Synostoses are to be met with under any of the above-mentioned circumstances, sometimes between single bones only, sometimes at several parts of a skeleton; and sometimes they are almost universal. Phœbus has recently seen and described an example of congenital synostosis, in which there had been a fusion of original points of ossification. There is a very similar case in the museum at Vienna, of congenital fusion of the second and third cervical vertebræ; only it obtains further importance from the fact, that the atlas is also congenitally united with the occiput. In another specimen the bones of the right forearm are continuous with the humerus, without the intervention of a joint. A similar synostosis, and one presenting considerable interest, is that of Nägele's obliquely narrowed pelvis, in which the sacrum is united with one of the innominata.

Anchylosis, in the restricted sense of extinction of a joint, especially that which is acquired, will be considered in the chapter on Joints.

Loosening of the natural connection of bones which are immovably articulated to each other, is denominated *Osteodiastasis:* when the same thing occurs between bones which move upon upon one another, the result is *dislocation*. In *diastasis* the change produced in the connecting medium depends upon circumstances: it is either stretched, attenuated and loosened in texture, or torn through. Gradually increasing extension leads to the first change, as is instanced by the cranial bones in cases of hydrocephalus, by the bones of the face, when stretched over fibrous polypi, or by the pelvic bones in parturition; while the second is caused by quickly acting force, and is exemplified by disjunction of the sutures when the skull is shattered, by the laceration of the synchondroses of the pelvis in very difficult parturition, or by the separation of the epiphyses in consequence of injury.

Moreover, the occurrence of diastasis is not only favored by previous disease, especially by inflammatory softening and loosening of the connecting substance, but it may be the immediate result of destructive suppuration of that substance, as is shown by the consequences which the pelvis sustains from the worse forms of puerperal disease.

The subject of dislocations will also be considered in the chapter on Joints.

§ 5. *Anomalies of consistence.*—These anomalies, expansion of the tissue, and so-called softening of bone, on the one hand, and induration on the other, are, essentially, changes of its texture, and of its chemical composition: the latter involve as well the mineral constituents as the animal basement, and they must therefore be treated of amongst the diseases of texture.

§ 6. *Solutions of continuity, and the process by which they are repaired.*—The solutions of continuity in the osseous system, which result from injury, present many varieties. Bones may be laid bare by the removal of their periosteum, and of the soft parts covering them: they may be wounded by more or less sharp penetrating instruments; from whence ensue the various kinds of punctured, incised, and shot wounds, by which the bone is either partly or completely perforated, as well as the wounds inflicted in operations, as amputation, trephining, in the removal of portions of bones, &c. They may be broken, and, as is well known, the fracture may be transverse, oblique, or longitudinal; there may be but one fracture, or several, in the same bone: and sometimes a bone is shivered, or crushed, and the fracture which results is comminuted.

Incomplete fractures or fissures are, for the most part, met with in the skull; as when only one of the compact tables is fractured, or when the inner table is disunited, while the outer remains uninjured.

There is a remarkable form of incomplete fracture, in which a bone becomes bent. It occurs on flat bones, like the skull, as well as on long bones. It is produced sometimes by sudden and violent mechanical force, sometimes by more gentle means, which act through a longer period either uniformly or with intermissions. Its occurrence is favored by the softness of the bones, which in fœtal life and in childhood exists naturally, but in subsequent periods of life is a morbid condition. These inflections of bone are chiefly observed in the skull of the new-born child as a consequence of the pressure which the head has undergone from the pelvis of the mother, or the forceps of the accoucheur: they may, however, be occasioned by accidental or intentional violence after birth; or they may take place in the bones of the limbs of persons who are affected with rickets or osteomalacia. They may be brought on by mechanical violence, or by excessive muscular contraction.

Any of these injuries may occur alone, or be combined in various ways with loss of substance: and further, they may be either simple, or complicated with considerable injury, bruising, and laceration of the soft parts, with shattering or splintering of the bone, with the presence of foreign bodies, with excessive degrees of inflammation of the soft parts, with gangrene, necrosis, &c.

In proceeding to the subject of the modes in which the most important injuries of bone are repaired, that by first intention, and that by way of suppuration, or second intention, I must refer, in order that the subject may be thoroughly understood, to what is said below on inflammation, suppuration, and necrosis of bone. The attention which has always been paid to the mode of union in cases of simple fracture has rendered that the foundation, as it were, upon which our ideas, as to the mode of repair in all other injuries of bone, have been based; and I, therefore, make it, both in its successful and unsuccessful issue, the subject of the following remarks.

Repair of fracture by the first intention.—When the extravasation produced by the fracture of the bone and simultaneous injury of the soft parts, and the vascularity of the soft parts, of the lacerated periosteum, the surrounding cellular tissue, and the adjoining muscles and their sheaths, have in some degree subsided, a reactionary process of inflammation is set up. The soft parts around the fracture, some of which have been injured by the same violence that produced the fracture, and others by the broken bone itself, become swollen and so blended as to constitute one uniform red, firm mass, infiltrated with inflammatory product, which encloses the broken part in a more or less smooth and round, or in an elongated, swelling. In the same manner, the medullary membrane becomes tumid and red; and, after a time, puts forth a red loose mass, which clings to the broken surfaces of the bone, but soon coalesces with the surrounding soft parts (Breschet's substantia intermedia). Thus each of the fractured ends lies in a sort of capsule of swollen soft parts. The innermost layer of the capsule is the periosteum, which, having separated from the surface of the bone to a greater or less distance from the fracture, is so intimately united with the surrounding soft parts as to be no longer distinguishable: a viscous reddish fluid exuding on its inner surface, fills up the space between it and the ends of the fragments.

Meanwhile, commencing at different distances from the seat of fracture, along the line where the periosteum remains connected with the bone, a reddish, semi-fluid, gelatinous substance exudes, which is greater in quantity the nearer the fracture: it is evidently connected more intimately with the bone than with the periosteum, but the former is entirely unaltered beneath it. Where it adjoins the bone it become cartilaginous, and subsequently ossifies; it then adheres intimately to the osseous surface, and if stripped off, leaves it perceptibly rough, and with its pores dilated.

This substance, as it increases in quantity, advances from the point at which the bone and periosteum are connected towards the seat of fracture, keeping close to the inner surface of the capsule of soft parts, and leaving a space between itself and the bare extremities of the bone, which is filled with the reddish viscid fluid before mentioned. At the same time, as it ossifies, it gradually assumes a more definite internal structure. If the fragments of the bone be in favorable position, the masses of callus, as they grow, assume a cylindrical shape, and arrive at the septum formed by the substantia intermedia. The septum has already acquired a cellulo-fibrous texture; but now it is gradually removed by absorption, and the masses of callus from the opposite ends of the bone coalesce over the fractured spot. What takes place outside the bone goes on also in the medullary cavity; a substance is effused, which everywhere ossifies and obliterates the cavity of the medulla. These changes constitute what is called the formation of early callus.

This callus gives some firmness to the fracture, and the time occupied in completing it—from thirty to forty days—forms, in a practical point of view, a very important period in the whole reparative process. It is named by Dupuytren the *provisional callus.*

Long before it has reached the state of completeness above described, a later growth, or formation of definitive callus, commences.

The fluid before spoken of as occupying the space between the broken ends of the bone and the callus (and a similar fluid which may be found also between the callus and the soft parts) becomes gradually more firm, receives vessels, and acquires a structure which at first resembles granulations, but at a later period is cellulo-fibrous. A small quantity of a reddish exudation appears beneath it on the denuded ends of the bone, and gradually unites with the granulation-like substance.

As a vascular communication is thus established between the surface of the bone and the surrounding soft parts, a formation of new osseous substance commences all round the fractured ends: it resembles the first formation of callus, but proceeds with less energy.

Both the earlier and the later callus increase in quantity, and at length unite together. They are, however, distinguished from each other by the difference of their texture, particularly of the softness of the second callus, when compared with the fully formed earlier growth. In this manner the broken extremities of the bone are surrounded by an uninterrupted osseous sheath which adheres to them both.

Last of all, in four or five months or more after the injury, the broken surfaces themselves unite within this sheath, though, indeed, the first traces of exuding osseous substance are sometimes perceptible much earlier, beneath the substantia intermedia, where it clings to the margins of the fracture. The substantia intermedia then disappears, and the fracture is completely repaired. The edges of the fracture are pretty frequently, though not always, distinctly rounded off.

During this consolidation, the remaining swelling of the soft parts subsides, and the medullary cavity begins to be restored at the fractured spot. The Haversian canals, in the mass of bone with which the cavity is filled, are gradually enlarged to such an extent as to render it cellular and areolar instead of solid; by further absorption it is entirely removed, and a new medullary cavity occupies its place. Sometimes a thin layer of the mass remains behind, united at the seat of fracture with the bony substance exuded from the broken margins, and for a long time obstructs the canal.

The callus, which was more or less uneven externally, has by this time become smooth. It is invested with a fibro-cellular membrane, which consists partly of the old periosteum of the bone thickened, and partly of the exudation poured out by the soft parts: the latter forms a new periosteum, unites into one membrane with the old one, and gradually becoming thinner, at last precisely resembles healthy periosteum.

Although the callus even when completely ossified, is at first clearly distinguishable from the old bone by the arrangement of its canals and lamellæ, yet in the end it acquires precisely the same characters as the bone. It is more than probable, that in process of time the callus diminishes in bulk, very gradually indeed, but yet so far as the relative position of the fragments of the bone, and its own original size, allow. Thus where simple transverse fractures have united, it shrinks so much as to form at last an inconsiderable elevation, which points out the original seat of fracture. But this is by no means to be regarded as an atrophy of the so-called provisional callus: no such decay, or involution, ever takes place, though it has been asserted by Dupuytren, and admitted by several others after him: there is no provisional callus, in the sense of a material for temporarily uniting or soldering the fragments together; and the reabsorption of the mass of bone which fills the medullary canal near a fracture, is to be regarded as one of the phenomena in the progress of the formation of a bone.

The firmness of the union is generally proportioned to the length of time that has elapsed since the occurrence of the fracture.

The process just described, in which bones reunite by first intention, is on the whole the same as that which takes place in injured soft parts. I have now to refer briefly to some points connected with the process, which have long been subjects of dispute; to describe what is observed when fractures unite under other, and especially under less favorable conditions than those which we have supposed in the foregoing delineation; and lastly, to treat of more important anomalies in the process, such as arrest, and the formation of new or false joints, repair by way of suppuration, and necrosis.

Long as the contrary opinion has been entertained, it is now beyond doubt, that in the formation of callus, no ossification of the periosteum takes place, any more than of the surrounding soft parts; but that, in the first formation of callus, the development of the whole osseous mass proceeds from the bone only is not fully ascertained, probable though it be. For, not to mention other facts, bone is sometimes found on the inner surface of periosteum; and the dura mater particularly, which is virtually a periosteum, frequently presents bony growths on its outer surface, which do not adhere to the bone at all, or do so very loosely, and only at a later period become closely connected with it.

During the whole process of forming callus, the old bone undergoes very trifling change; and it requires close examination after the removal of the exudation, to discover that the pores on its surface are somewhat enlarged, and that the surface itself is rather rough, in consequence of some of the new matter remaining adherent to it. There is no greater change even when the exudation has ossified. When the formation of callus is complete, and the fragments have reunited, the bone sometimes shrinks, and becomes palpably thinner, and its cavity smaller than before.

When only one of two adjoining bones is broken, as in the forearm or leg, the uninjured one takes part in the process of forming callus. Osseous matter is exuded by it near the fracture, which unites with the callus of the broken bone; for just as the substantia intermedia disappears when the broken surfaces of the bone unite, so the periosteum covering the callus of the uninjured bone is absorbed, and the two growths of callus coalesce. But still more frequently is this the case when both bones of the forearm or leg are broken, or whenever there is a fracture of two or more adjoining bones, such as the ribs. In fractures in the neighborhood of joints, fears may even be entertained, lest union between the articular ends of the bones be occasioned by the callus.

Long oblique fractures involve a mass of soft parts in the reactionary process, and are attended by the formation of a quantity of callus, proportioned to the extent of the fractured surfaces. So, too, the greater the displacement of the fragments, the greater and at the same time the more misshapen, will be the swelling of the soft parts, and the subsequent callus. The same occurs when the fracture is comminuted.

The great extent to which fragments, when displaced, are denuded occasions a difficulty in the formation of the secondary callus, and retards the process of repair; but in all essential particulars the process is the same. The callus encloses the fragments, and generally forms a bulky mass, which connects the opposite surfaces of the bone together. The medullary cavities, at first open, become blocked up with callus, which unites intimately with the swollen soft parts around; but gradually opening again, they become continuous with a new medullary cavity, which forms in the callus. Sometimes even when contiguous walls of the bone are firmly consolidated together, they and the intervening callus are absorbed, and the continuity of the original tube is restored by means of a new transverse or oblique canal. A remarkable analogy to the mode in which bone is originally developed is observed in the universal tendency to form medullary cavities in the interior of all large masses of callus, as in exostosis, and the more bulky osteophytes.

Separate fragments unite within the callous enlargement in more or less favorable positions.

The surface of large masses of callus is marked with grooves in which tendons or large vessels lie; and sometimes they are even perforated in various directions by small canals which those structures traverse.

Of arrests of the growth of callus in general, and of new joints in particular.—The modes and degrees in which the formation of callus may be arrested, are very various. The quantity formed may be insufficient for its purpose, or there may be none at all: it may undergo the change which is incident to it very tardily, and not be ossified till a very late period; or may be imperfectly, or but partially, ossified: or the exudation, instead of becoming cartilaginous and bony, may assume and retain an apparently ligamentous structure. Such arrests may take place on both fragments, or predominate on one: and further, the anomaly may extend to both the first and second growths of callus, but usually it occurs in the latter only. In such cases, days and even weeks after the occurrence of a simple fracture, neither the bone nor the soft parts around it exhibit well-marked, or indeed any traces of reaction; and if the fracture should at last unite, it does so by the way of suppuration. In other cases, the growth of callus may be insufficient, the masses first formed may meet each other only at a few points, or may even not meet at all; as is the case when there is great displacement of the fragments. The secondary callus may not unite completely with the primary, and may remain soft and cartilaginous, or there may be too little of it formed, especially at the fractured extremities of the bone; in which case, its metamorphosis may be arrested or anomalous; or there may be no callus formed at that part at all.

It is in the last-mentioned circumstances that the cause is found for the formation of new, or false joints. (Articulus novus, spurius, præternaturalis; pseudarthrosis; articulation surnuméraire of Béclard.)

The unnatural joints, which result from fracture, are of two different kinds: one more or less resembles a synchondrosis; the other is like a diarthrosis, and is accordingly, in its proper sense, a new joint.

In the former case, the fractured ends of the bone are held together by a ligamentous tissue. Either a disc of ligament, the thickness of which may vary, is interposed between them, and allows of but little movement; or, as occurs when there has been loss of substance, either from injury, from considerable absorption of the fractured ends, or otherwise, ligamentous bands connect the fragments, and allow them to move freely on each other. The connecting substance appears to be nothing but the substantia intermedia mentioned above, which, as the formation of the secondary callus has failed, or been insufficient, remains in its first state.

In the second case, a ligamentous articular capsule is formed, and is lined by a smooth membrane, which secretes synovia: the fractured surfaces adapt themselves to each other, and become covered with a layer of tissue, which is fibro-ligamentous, or more or less fibro-cartilaginous, or which resembles, and sometimes (Howship) really is, cartilage: they may articulate immediately with one another, or may have between them an intervening layer of ligament, which corresponds to an interarticular cartilage; and their movement upon each other is more or less free, according to the size of the articular capsule, and the form of the articulating surfaces. These last are sometimes horizontal and smooth; they glide over each other, and allow of restricted motion: sometimes one surface becomes convex, and the other concave: sometimes both are rounded off, and lying within a capacious articular capsule far apart, they come in contact only during particular movements. The articular capsule is the product of the inflammation of the soft parts: the cartilaginiform layer, which covers the ends of the bone, is secondary callus arrested in its metamorphosis and converted into a fibroid tissue: the other ligamentous cords, which are sometimes present, and the structures resembling an interarticular cartilage, are remnants of the substantia intermedia.

Both forms of new joint, but more particularly the synchondrosal form, have an analogue in the lateral new joints sometimes found between the masses of callus which are thrown out around two adjoining fractured bones. In the forearm and leg, and between the ribs, for instance, new joints are sometimes met with between the masses of callus after fractures have united.

Between that kind of new joint which constitutes a firm synchondrosis, and that which as nearly as possible resembles a natural diarthrosis, there are numerous gradations.

The circumstances which arrest the growth of callus generally, and give rise to the formation of false joints, are as follow: advanced age and senile atrophy of the bones; emaciation in consequence of disease, or loss of the fluids; cachexia generally; diseases of the bones in particular, such as rickets, osteomalacia, too severe inflammation, suppuration, caries, and necrosis at the broken spot; paralysis, and similar affections (on which subject reference may be made to Roechling's experiments on animals); pregnancy; any improper bandage which stops the access of the fluids to the part (a circumstance which is explained by Brodie's experiments of tying the crural artery in animals, in which the femur was broken); inquietude of the limb; considerable displacement of the fragments, and the occurrence of the fracture within the capsule of a joint.

But these conditions do not always put forth their power of arresting the formation of callus and the repair of fractures; certainly they do not always act equally. Thus the fractures of bones affected with rickets or osteomalacia are not very unfrequently united by a mass of callus, which not only is sufficient to repair the fracture, but even has a more perfect internal structure than the other bones: fractures very often unite at every period of pregnancy; and the neglect of quietude is unquestionably rather a grave impediment to the reunion of fragments in proper position than to the formation of callus and the repair of a fracture generally.

Fractures within the capsule of a joint require more particular notice. It is a fact ascertained from much experience, and now established as a rule, that fractures within the capsules of joints very rarely unite completely: the fragments become bound either firmly or loosely together by a ligamentous apparatus; or, their surfaces becoming eburnated or covered with membrane, they form an articulation with each other within the old joint. Many reasons have been assigned for the great deficiency in the formation of primary callus around these fractures, as well as for the arrest of the growth of secondary callus; but which of them is to be regarded as the true and the universally applicable one is not ascertained. As fracture of the neck of the femur is the principal instance of the kind, and is that which has led to all the investigations that have been instituted on the subject, I shall enter more particularly into the consideration of it when treating of Diseases of the Bones of the Lower Extremities.

Union of fractures by suppuration.—Compound fractures unite in a different manner from that by first intention: yet the repair by suppurative inflammation, in its essential particulars, has been far too little investigated. It is analogous on the whole to the repair by first intention, certain stages of the latter always occurring in the course of it; only, as must be obvious, the repair is effected with more difficulty and at a later period, and sometimes is never completed.

In this as in the other process, a capsule of soft parts forms around the broken ends of the bone, but the inflammatory product contained within it is pus, just as in any wound which may exist at the same time in the soft parts, and the capsule being lined with a grayish-red, translucid, jelly-like, granulating layer, which is covered with pus, is in fact a closed abscess.

The early callus, as in union by first intention, appears as a gelatinous exudation, which subsequently becomes a cartilaginous, and then a bony stratum. It springs from the bone, at the part which has remained covered by periosteum; and then, keeping off from the denuded ends of the bone and from the fracture itself, it advances on the inner wall of the capsule towards the broken part, the ossifying cartilage being developed from the granulating layer.

The broken ends of the bone, so far as they are denuded of their periosteum, are washed in the pus: they lose their natural color, and look bleached and dull white, but are otherwise unchanged—even at the surfaces and margins of the fracture.

It is not usual to see any plastic exudation in the open medullary cavity: the fractured surfaces are everywhere washed in the pus: the internal surface of the bone—that which faces the medullary cavity—appears dull white and dead to a greater or less depth on one side, or for the greater part, or the whole, of its circumference: the cells and spongy tissue are in the same state; and the medulla at that part is collapsed, soft, and discolored, and is dissolving in the pus. Beyond the confines of this change, however, the medulla is swollen and reddened, and if the necrosis have not extended all round the bone, it protrudes, as it does in the process of union by first intention, beyond the fractured surfaces.

Now, it is more particularly the growth of secondary callus which is late in commencing, which very often suffers more or less considerable interruptions in its progress, and which differs most from that which is formed in union by first intention. The ends of the bone being washed in pus, and thus kept in contact with a fluid incapable of organization, die: and the extent of the necrosis, whether it shall be superficial, shall be confined to the inner layer of the bone, or involve its whole thickness, is unquestionably determined by the extent to which the periosteum is stripped off, and the medullary membrane destroyed. As soon as the necrosed part has exfoliated, granulations appear on the bare surfaces and margins of the fragments, and become the basement in which the new bony substance, or secondary callus, is deposited.

It is by the very slow manner in which the exfoliation of the necrosed bone takes place, that the formation of the secondary callus is delayed: and as not unfrequently the powers of the system are almost exhausted before it is completely formed, it is often produced in insufficient quantity, or arrested in its perfect internal development, that is, in its ossification. Moreover, as soon as the exfoliation and entire removal of the necrosed piece are effected, the inflammation ceases, and a great part of the granulations which were exuded for the purpose of producing new bone, goes to form on all sides a cellulo-fibrous, ligamentous, and cicatrix tissue: hence it is, that the repair of fractures in this manner is so often incomplete, and is attended with so extensive and permanent a loss of substance. Whatever the condition of the early callus, whether it be abundant in quantity and thoroughly organized, or otherwise, the granulations supply an inadequate substitute for the bony substance which has been lost by exfoliation: instead of changing into cartilage and bone, they become converted into fibroid tissue; and thus the repair which ensues is attended with shortening, with disfiguring cicatrices, or with an artificial joint.

Repair of the bendings and fissures of soft bones.—When the bones of children or of persons affected with rickets have been slightly and gradually bent, and the bony tissue and periosteum have been stretched without suffering a breach of their continuity, they are gradually restored to their natural direction without giving any sign of reaction. And those injuries also in which the bones of children are rapidly and violently bent, are easily repaired. But when bones are bent to an angle by a greater and more sudden force, and a real, though not always perceptible, solution of continuity takes place, whether it be on the one side a tearing asunder, or on the other a crushing, of the outermost layers of the bony tissue, the injury is repaired like a fracture by first intention. When bones affected with rickets and osteomalacia are bent in this manner the callus generally continues in a soft half-cartilaginous state, and does not obtain its perfect internal structure till the disease is cured. To this, however, there are exceptions, for bones affected with these diseases, when bent and partly fractured, and also when broken quite through, are sometimes reunited by bony callus: and it is not till after it has reached a certain stage of development, especially in osteomalacia, that the callus undergoes the peculiar metamorphosis.

Repair of injuries of bone complicated with loss of substance.—The mode in which wounds of bone combined with loss of substance are repaired is, on the whole, the same as that by which fractures unite. Under favorable circumstances it is effected by the first intention, and the osseous mass exuded from the surfaces of the wound in the bone serves not merely to reunite the bone, but also to supply the place of the part which has been lost. This is true of loss of substance as well of one bone as of another, and in whatever degree, or in whatever form it may have occurred. Irrespectively of unfavorable general conditions, it may be said that the greater the loss of substance, and the more the repair proceeds by way of suppuration and granulation, the less complete will be the reproduction.

Hence it happens that injuries of this kind are followed by permanent loss of more or less of the substance of the bone, and that in long bones shortening, or a false joint is produced.

Wounds of the skull made with a trephine are extremely seldom closed by bony substance altogether, but the circumference of the opening mostly exhibits a growth of bone which may be compared to the two formations of callus. It proceeds from the surfaces of the compact tables as well as from the surfaces and margins of the wound; but is insufficient to close the opening, and the defect is, and continues to be, for the most part supplied by a ligamentous (fibroid) plate, which adheres closely to the dura mater on the one side, and to the pericranium on the other; the trifling amount of (secondary) callus which proceeds from the surfaces of the wound juts into the substance of this plate.

The adjoining part of the wall of the skull is not unfrequently at the same time considerably attenuated, so that the fibroid layer which closes the opening is continuous with a margin of bone, towards which both surfaces of the skull are bevelled off.

In some cases true bone is developed in this fibroid plate. It assumes the form of needles and small plates, and gradually becoming identified with the callus which is growing inwards from the margin, it at length effectuates the closure of the opening by bone. Similar bony growths are met with in false joints in the ligamentous structures by which the fragments are connected together.

The way in which the wounds of bone made in amputations are healed accords with what has been described above: the medullary canal closes, the stump becomes rounded off, and unites with the soft parts and their cicatrix by an intervening cellulo-fibrous tissue, which supplies the place of a periosteum. If the inflammatory process should lead to suppuration in the bone, and still more, if suppuration take place in the periosteum and medulla, necrosis ensues to a corresponding extent; and when the dead piece has exfoliated, the cure is effected by way of granulation.

Under such circumstances, the condition of the stump is sometimes unfavorable: the callus may be insufficient in quantity and density, and therefore incapable of enough resistance: the stump may be attenuated and end in a point; or, on the contrary, the callus may grow from it exuberantly in the form of some of the various osteophytes.

Repair of injuries in which bone is denuded of its soft coverings.—There is no question that injuries of this kind are repaired by first intention. The soft tissues and the bone together furnish an exudation which becomes organized at one part into a layer of callus and at another into a cellular or cellulo-fibrous tissue; and it will be observed, that the connection between the two new products is considerably closer than that which exists between bone and its periosteum in their normal condition. But in the unfavorable circumstances under which these accidents occur, and in which they remain, for a more or less lengthened period, until the arrival of surgical aid, such injuries are more frequently repaired by suppuration and granulation, after the exposed layer of the bone has exfoliated. Not unfrequently, indeed, they lead to a fatal termination, by the extensive suppuration in the soft parts, and the necrosis of the bone, which, like other injuries of bone complicated with wounds of the soft parts, they set up.

§ 7. *Diseases of Texture.*—Although diseases of bone generally, and those of its texture especially, have been the object of much valuable investigation, both clinically and anatomically, yet our knowledge of them is still very defective; and perhaps nowhere amongst the diseases of the solid organs is the need of chemical research keeping pace with anatomical inquiry more perceptible than in the subject now before us. And another circumstance, which renders it extremely difficult for the pathological anatomist to deduce his single results from numerous investigations, is the want of accurately distinguishing between different affections of bone, according to their local characters, as well as to the general symptoms, on the living patient; for regard is usually confined to syphilis, scrofula, and particularly to gout, when subsequent anatomical examination discloses changes of an entirely different nature.

1. *Congestion of bone, Hemorrhage.*—Bones, like every kind of soft part, are subject to congestion, though, on account, probably, of the little attention which is generally paid to them in examinations after death, changes of their vascular condition are seldom noticed and estimated. Congestion is most frequently observed in the bones of the skull, the vertebral column, and the spongy articular extremities of long bones. In new-born children, and during childhood, considerable congestions of the cranial bones are met with: passive and mechanical congestions of the vertebræ, especially of the lumbar portion of the column, occur, even in advanced life, when the circulation through the ascending cava and vena portæ is impeded: and cases of osteoporosis, rickets, &c., are

accompanied by extreme congestion. There is no question that habitual congestions not unfrequently lead to hypertrophy of bone, especially in the form of induration: in cases of atrophy, where congestion coexists with expansion of bone, that is, with enlargement of its Haversian canals and cells, it may be produced by the wasting of the tissue of the bone.

Hemorrhage takes place from bone under various circumstances. The vessels of bone, periosteum, and medullary membrane pour out their blood when torn in the various injuries which happen to them. Occasionally very considerable bleeding takes place under the periosteum, in the spongy tissue, or in the medullary cavity, from the exposure and corrosion of vessels of various sizes, by caries. But the most interesting cases are those in which spontaneous hemorrhage, resulting from extreme congestion, originates from the delicate vessels that pass between periosteum and bone, and ramify in the grooves or half canals on the surface of the latter. The principal instance is that met with on the cranial bones of the new-born child, which is known by the name of Cephalhæmatoma. I shall have to advert to it again amongst the Diseases of the Bones of the Skull, where it can be more conveniently described.

2. *Inflammation of bone.*—Inflammation of bone (ostitis) is sometimes evidently the result of external causes, of various injuries, for example, most of which have been already enumerated, of concussion of the bone, or of cold; sometimes it arises from internal conditions, as when some constitutional affection, whether syphilis, the mercurial cachexia, scurvy, gout, and the like, or an exanthematous process, fixes itself in a bone. But the etiology of inflammation of bone is a subject which requires, more than many others, to be cleared up.

Moreover inflammation of bone is frequently a secondary disease propagated from neighboring tissues, especially from the periosteum.

It is sometimes an acute inflammation, especially when produced by external injury; very frequently it is chronic, and is almost always so when it arises from any constitutional affection. It is sometimes confined to one bone, or to one circumscribed spot on a bone; sometimes it attacks several bones, or most, or nearly all of them, not perhaps all at once, but one after another in more or less rapid succession. At one time it affects the outer layer of a bone, and is combined with periostitis; at another the inner strata, when it is associated with inflammation of the medullary membrane: the accompanying inflammation of the periosteum and medullary membrane may be an original part of the inflammation, or a later addition to it. Lastly, there is a third case, in which a bone is inflamed in its whole thickness: in some instances it is so from the first, in others the inflammation reaches that extent later in its course.

It is situated sometimes in compact substance, in the compact portion of a bone; sometimes in spongy substance, in spongy bones, and spongy parts of bones; and, external causes being excluded from consideration, the selection of its seat depends in a remarkable manner on the constitutional affection which gives rise to it, a peculiar preference being manifested sometimes for one portion of the skeleton, sometimes for another, and sometimes for particular bones. Inflammations of bone vary much, and, in a practical point of view, materially in the degree and nature of the inflammatory process; and exhibit it first in the composition of their product (the exudation), and then in their consequent terminations. It would be inconsistent with nature, and in fact impossible, in an anatomical delineation of inflammation of bone, to make a broad division of it into acute and chronic; I shall, therefore, be careful only at the proper points to mark the transitions of one into the other, and the characteristics by which they are distinguished.

A very moderate degree of inflammation, in the outer lamella of a bone, for instance, produces a gelatinous, dark-red exudation, which gradually changes its color to bluish-red, yellowish-red, and reddish-white, and at length becomes quite white: at the same time passing from its original gelatinous condition, it forms a coagulum like white of egg, then becomes a soft, flexible cartilage, and, finally, reddish-white, succulent bone. In this state it invests the bone, and constitutes, according to its quantity, either a white, porous, and scarcely perceptible film, or a thicker layer, that resembles fine felt or velvet. The periosteum appears at first injected, bluish-red, infiltrated, and decidedly swollen, and generally has but a loose connection with the exudation; for the latter, especially after it has ossified, adheres somewhat closely to the bone: sometimes, however, when the periosteum is peeled off, part of the exudation comes away with it. There is no change in the bone till the process is advanced, and then its grooves and the pores for its vessels are manifestly widened. The ossified exudation afterwards unites with the surface of the bone, and either forms uninterruptedly an addition to the thickness of the compact wall, or is connected with it by a layer which remains spongy (diploetic). In some of the thicker layers of bony exudation, occasional voids of various dimensions are met with, which are filled with a vascular tissue containing medulla: they may, on the macerated bone, be easily taken for the losses of substance produced by caries; but without doubt they are merely the result of absorption in bone already formed, and are analogous to the formation of cancellous tissue, and a medullary cavity in callus. (Compare with this and with the following, what has been said on the subject of the osteophytes.)

The principal opportunities of seeing this process are obtained in the neighborhood of more intense spots of inflammation, and around caries.

The inflammation may recur in the exudation at any period of its ossification, and lead to a corresponding increase in the size of the bone.

There are other inflammatory processes, some of them more acute, which return from time to time, and appear to be of a specific nature. Like those already described, they extend sometimes to several bones, and they deposit abundant exudations, which may assume the form mentioned above, or the various other shapes delineated in the section on the osteophytes: the periosteum, at the same time, becoming hypertrophied, acquires a fibro-lardaceous, callous structure, and sometimes an enormous thickness.

An inflammation of this kind may be often observed beneath chronic ulcers on the inner surface of the tibiæ. The periosteum and adjoining cellular tisue, having been converted into a lardaceo-callous substance, form the base of the ulcer, and cover a luxuriant growth of curled bony plates, like madrepore, which are arranged perpendicularly upon the bone: the periosteum sends processes between, and forms sheaths around the separate plates. A villous, or a spavined and laminated, osteophyte is usually seen in the neighborhood, and hence, as the new tissue becomes indurated, a circumscribed portion of the bone is increased in bulk.

When the inflammation is seated in the inner lamella of a long bone, or in the diploetic substance, it pours forth its product on the inner surface of the medullary tube, or on the walls of the cells of the spongy substance; and the tube becomes narrowed, and the spongy substance condensed. The process of exudation very often occurs on both the outer and the inner lamella of a bone together; and sometimes the intermediate substance also shares in the process, and the ossifying exudation, deposited on the walls of the Haversian canals, produces induration (sclerosis) of the bone.

Besides these there are, no doubt, inflammations of bone, especially such as are slight in degree, and chronic in their course, which give rise to products that become organized in various other ways. Some change into osseous substance, the texture of which deviates from that of healthy bone; while others form fibroid, or cellular tissue, or a substance which resembles the jelly of spongy bones. Such products occasion a loosening and expansion of the bone proportioned to their quantity; and many of the osteoporoses, which are attended with increase of the volume of the bone, are, no doubt, due to such processes of inflammation, or as they are called, irritation.

A high degree of inflammation in bone leads to the effusion either of a fibrinous product, which more or less rapidly softens; or of a purulent exudation, which varies in fluidity, according to the quantity of serum it contains, and is yellow and frequently tinged by an admixture of the coloring matter of the blood, and of blood itself; or, lastly, of a greenish or brownish discolored sanies. There are some striking instances of this kind of inflammation, which run their course very rapidly: they occur not only after injury of a bone, especially after concussion, but also in consequence of cold; and they are associated with endocarditis and several other exudative inflammatory processes. The periosteum in these cases is loose and movable to some extent over the bone, the exudation being poured out beneath it: in well-marked cases, it becomes loose all round the bone, and distended into a fluctuating sac, which contains a large quantity of the exudation. The tissue of the periosteum is loosened and infiltrated: the bone is of the same color as the exudation, and has accordingly a dun, a dirty yellowish or a greenish or reddish appearance, which results from all its spaces being occupied by the exudation. The exudation is deposited in greatest quantity in the cancellous structure; but when the compact tissue is minutely examined, the Haversian canals are found to contain it too. The surface of the bone, especially when the exudation is sanious, appears rough, that is to say, its outermost layer is eroded, and the loss of substance is produced by the solution of its tissue during the process of exudation itself, and by contact with a product which exerts an absolute dissolving power. The walls of the Haversian canals exhibit similar loss of substance, and are rough and eroded, or completely perforated. The cells of the cancellous structure show it still more clearly, and the membrane which covers its cells and network is opaque, dull, and discolored like the exudation, and is easily torn. It is this form of ostitis which, if it do not prove fatal by its coexistence with other exudative processes, very commonly puts life in the greatest danger, or actually destroys it by leading to absorption of the purulent and sanious matter into the circulation (the coats of the veins probably being dissolved in the exudation around them), and by metastasis. When the case is favorable, necrosis of the diseased bone, or portion of bone, is inevitable.

But inflammations of bone, which are accompanied by the production of pus and sanies, are more frequently chronic. The suppurative inflammation mostly occurs as a consequence of complicated injuries of bone and of necrosis: the chronic inflammation with an ichorous product, arises from internal causes, sometimes commencing spontaneously, sometimes being excited by injury to the bone, and sometimes, under the influence of the internal causes just alluded to, being an early or a late degeneration of the suppurative inflammation. It produces loss of the substance of the bone by ulceration, and constitutes Caries (Vereiterung—Beinfrass).

In suppurative inflammation, especially in cases of considerable injury of bone, exposure, for instance, for a lengthened period, there very commonly takes place, in the benumbed tissue, a visible exfoliation of a layer of bone of various thickness. When the necrosed portion is very thin, exfoliation is rightly assumed to be going on imperceptibly by the separation of small and scarcely discernible particles. But there are several cases in which symptoms have been ascribed to necrosis and insensible exfoliation, merely as it appears, for the sake of the theory, and in which no such process occurred. The circumstances were in reality more favorable; for as, when suppuration in its most benignant form takes place anywhere, the first secretion of pus is attended by some breach of substance—by some solution—of the tissue in which it occurs, so is it in these cases. The softening which many observers have adduced as a constant phenomenon in the suppuration of a layer of bone, must be regarded as such a process of solution: a portion of bone disappears, and the exudation beneath it, like that beneath exfoliated bone, becomes organized into granulations, which spring from the tissues lining the Haversian canals. This view is supported generally by the process of sanious destruction, which is allied to the suppurative, and is only distinguished from it by the relatively greater amount and the progressive increase of the solution; while it is upheld more particularly by the state in which a bone is found when this peculiar loss of its substance is the consequence of an acute inflammation attended by the production of pus or sanies, or when it results from caries: and lastly, it is further borne out by the condition of a sequestrum: but this is a subject to which I shall revert when speaking of necrosis.

A spot of suppuration in bone is always skirted by an inflammatory process, which leads to an exudation of bone. If the affected part be the outer layer, an osseous exudation is found not only encircling the suppurating spot, but also on the inner table of the bone, and *vice versâ.* When suppuration takes place in spongy bones, this process occasions so much condensation, that in a few cases an abscess becomes enclosed within a capsule of compact bone. This capsule is lined by a cellular membrane which is richly supplied with vessels, and it resembles an abscess in the soft parts enclosed within callous walls. Abscesses of this kind have been pretty frequently observed in the extremities of the tibia (Brodie, Mayo); and on a few occasions they have been met with in the compact substance of the shaft of that bone and of the femur (Arnott).

Having hitherto treated of those terminations of inflammation in which bone is increased in volume and density (sclerosis, which is equivalent to induration in the soft parts), and in which suppuration ensues; I am induced by the importance of the subjects to bestow separate sections upon the consideration of caries—chronic inflammation with production of sanies—and necrosis.

3. *Ulceration of bone,—Caries.*—This disease corresponds to ulceration in the soft parts. It is sometimes the immediate result of an inflammatory process of low type (dyscrasia), the product of which exerts a solving power upon the bony tissue: the scrofulous and syphilitic ulcerations of bone are of this kind. Sometimes it arises out of simple suppuration in a bone in consequence of local or general (internal) causes. It is, moreover, frequently set up by ulcerative processes going on in adjoining soft parts; a frequent instance of which is presented by the caries of the articular ends of a bone, which ensues upon disorganization (Verjauchung) of the soft tissues of a joint. Lastly, it results also from the softening and ulcerative inflammation of morbid growths in bone, such as tubercle, cancer, &c.

Caries is sometimes situated at the surface (caries superficialis, peripherica), and sometimes originates in the interior of a bone—in its medulla (caries centralis, profunda). In its extent it may be total or partial: it may involve a whole bone, as one or more of the vertebræ, of the carpal or tarsal bones, or the whole of a finger or toe; or it may attack a portion only of one of the larger bones, such as the end of one of the long bones composing a joint, or a circumscribed spot on the shaft of a bone.

Though it chiefly affects spongy bones and parts of bones, it is not altogether rare in the compact tissue; indeed, certain forms of dyscrasia establish their ulcerative inflammation by preference in that tissue: generally speaking, we may say that there is no bone which may not be the seat of the disease. It is most frequently met with in young persons as a scrofulous affection.

It may come on whether the tissue of the bone in which it occurs be in its original healthy state, or have been previously diseased; it may occur, for instance, in a rickety bone.

Its course is generally chronic; but in the extensive devastations which it commits, and the fatal exhaustion which it sometimes produces within a short period, it frequently exhibits the character of an acute disease. It often threatens life, moreover, less by its own progress than by exciting inflammation in neighboring important organs; it does so, for example, when it occurs in the skull.

An ulcer in bone presents numerous varieties corresponding with the kind of constitutional affection which gave rise to it. I shall, however, first treat of the process of ulceration in bone generally, without reference to its varieties, as there will be an opportunity in another part of detailing the characters and differences which the ulcer obtains from the several processes of dyscrasia in which it originates.

The appearances presented by an ulcerated bone when examined in the recent state, vary according to the progress which the disease has made; and in every stage of the affection its characters are far better marked in a spongy than in a compact bone.

When caries is superficial, the compact bone is found covered with ichor, and rough, as if it had been gnawed: this appearance is given by the unequal loss of substance which the outward lamellæ have sustained. The Haversian canals are enlarged, but not uniformly: the tissues contained within them form in part a disorganized soft and shreddy mass, infiltrated with ichor; or spongy granulations which easily bleed, grow from them luxuriantly, and advance outwards over the rough surface of the bone, whilst internally they partially or completely fill the enlarged Haversian canals. In both cases the bone appears porous or cancellous, but its color differs in the two: in the former, it is discolored by the contents of the Haversian canals; in the latter, it obtains various tints of red from the color of the granulations.

When caries affects cancellous tissue, the bone acquires a livid red color, especially if the granulations be at all abundant; it becomes soft, resembles a mass of flesh traversed by a delicate and brittle bony skeleton, and is easily cut with a knife, or yields to light pressure with the finger: lastly, it becomes swollen.

In cases of central caries, the swelling sometimes produces expansion of the bone, especially if it be a spongy bone, for the thin wall gives way and becomes distended.

The loss of substance which the bone sustains is occasioned by its solution in the sanious product which is effused by the inflammation into the Haversian canals. These canals enlarge in all directions, though not uniformly, and contain in different proportions, on the one hand, sanies and the soft parts which naturally fill them, discolored and disorganized (verjaucht); and on the other hand, granulations. It is thought by Delpech and Berard, Pouget and Sanson, and by Mouret, that a peculiar fatty matter is generated in carious bones; Mouret differs from the others, however, in believing that the organic principle (the gelatinous portion) does not disappear from the bone.

The sanies produced by the bone is an acrid fatty fluid, itself discolored in various ways, and which, as is well known, blackens silver probes and linen. It almost always contains small particles of bone, discolored and brittle, which look as if they had been calcined, and are, in fact, loosened remains of the bony tissue, which is being destroyed. They are, without doubt, minute portions of necrosed bone; for in every form of caries, small imperceptible particles of bone die and are cast off. More rarely it happens, that necrosis of a larger piece of the ulcerating bone takes place (caries necrotica). In that case the portions of bone die sometimes without partaking at all in the inflammatory process, and simply from the access of their fluids being cut off by the carious destruction which is going on around them; and sometimes they die from the inflammation and disorganization.

Whilst this disorganizing process (Jauchung) is going on in the bone, more or less of the adjoining osseous tissue and soft parts are always inflamed to a greater or less distance. The inflammation is sometimes chronic, and the soft parts become infiltrated with a gelatinous or gelatinous and lardaceous product, and indurated; at other times it is acute, and leads to suppuration and ulceration. The periosteum, and the ligamentous tissues connected with the bone, are, of course, involved in this change in the soft parts. The mode in which the ulcer of the bone opens externally, varies according to circumstances: sometimes one large abscess is formed; at other times, one or more straight or tortuous, single or branching, long canals (fistulæ, sinuses), either lead directly outwards, or not unfrequently pass to very remote distances; the orifices of the sinuses are usually marked by rather a hard margin, which surrounds them like a rampart.

The carious bone, when macerated and dried, looks rough, and as if corroded: from being perforated in various ways by the unequally-enlarged Haversian canals, it has a spongy, porous, worm-eaten appearance; the cells of its cancellous structure are enlarged; its walls and network are attenuated or demolished; and hence it is lighter than natural, discolored, expanded, and very brittle.

New osseous substance, which assumes the form of some of the different osteophytes, is sometimes deposited around the ulcerated spot, both on the surface of the bone, in its medullary canals, and in the cells of its spongy substance. And bone is deposited not only on the diseased bone, but on others also which are near it.

In other cases the neighboring bones are found in a state of rarefaction (osteoporosis), of areolar expansion, combined with hypertrophy, or inflammation of the soft parts of the bone, and, at length, of atrophy of their tissue.

Caries will heal, even in cases where it has committed great devastations, by a change of the ulcerative into a healthy suppurating and granulating process. The subsequent reproduction of bony substance is small in proportion as the amount of destruction has been great, and hence there will be more or less deformity, as well as variety of size, in the cicatrix.

Caries, as has been partly mentioned already, and will also be further pointed out hereafter, must be carefully distinguished from several other losses of substance in bone.

4. *Necrosis* (*mortification of bone*).—Necrosis in bone corresponds to mortification in the soft parts, more particularly to dry gangrene, or mummification. It has in general a less serious character than the latter, inasmuch as by the application of appropriate artificial remedies, it very commonly, though slowly, gets well; and is only fatal in the few cases in which the strength of the patient is exhausted by the excessive secretion of matter that takes place, for the purpose of removing the dead piece of bone. Necrosis sometimes arises from external causes, such as injuries of various kinds, by which the bone is shaken, crushed, or laid

bare, from the influence of severe cold, heat, &c.; and sometimes it is developed, as is said, from internal causes, amongst which various kinds of constitutional disorder are enumerated, especially scrofula, syphilis, the state of constitution resulting from the abuse of mercury, gout, scurvy, &c., and the cachectic state succeeding acute exanthemata, especially variola and measles. An internal cause of this kind, in producing necrosis, may do so, not only by exciting inflammation and suppuration of the periosteum or medullary membrane, but also, as is extremely probable from analogy, by leading to inflammation of the bone.

Necrosis may affect either the whole of a bone (necrosis totalis), or, which is more frequent, only a part of one (necrosis partialis). In the latter case the outer lamella may be alone involved (necrosis externa superficialis), or only the inner layer of a cylindrical bone, or diploetic substance of a broad bone (necrosis interna, centralis); or again the whole thickness of a bone, within certain limits, may be necrosed. But it is very rarely that a necrosed piece admits of its whole extent being so accurately defined; that which at one part includes the whole thickness of a bone, runs out at its extremity into a superficial necrosis. Internal necrosis, too, occupies a bone very unequally, and at some parts frequently extends nearly through its whole thickness.

Moreover, the boundaries of a piece of dead bone are irregular in every direction; its margins are notched and sinuous; and its thickness, especially if the piece be peripheral or central, is very unequal in different parts.

The less vascular compact bones are those which are chiefly liable to necrosis; and of such the shafts of the long bones, more particularly the tibia, and after it the femur, humerus, ulna, radius, and fibula, and the bones of the skull, are most affected. Unlike caries, it rarely occurs in spongy tissue. Necrosis of the long bones very commonly terminates at their cancellous articular extremity, or at the junction of the epiphysis with the shaft. Every period of life is subject to it, though it is most frequent in young persons; and in them it occurs as scrofulous necrosis, or necrosis based upon a scrofulous constitution.

Necrosis sets up an active inflammation in the adjoining healthy bone and surrounding soft parts, which goes on to suppuration, and continues until the dead piece is removed either by nature or art. Separation in the former manner is very seldom completed, for the suppuration rather exhausts and destroys the patient. The matter discharges itself externally by one or, more commonly, by several ulcerated sinuses.

The purpose designed in the suppurative inflammation is to separate, and finally to throw off, the dead portion of bone, which then obtains the name of *sequestrum*. The necrosed piece is at first distinguishable, to a certain extent, from healthy bone by its bleached and somewhat discolored appearance, but its boundaries are at that time indistinct, inasmuch as the discoloration of the dead part blends gradually with the color of the healthy bone.

All around the necrosed portion, that is to say, at its margins, and at the part where its surface is opposed to that of the healthy bone, the latter undergoes a gradual expansion or rarefaction of its tissue by the enlargement of its Haversian canals, assumes a rosy color, and becomes succulent. It acquires gradually an areolar structure, and is thus more rarefied: at length it disappears altogether, and a red soft spongy substance, a layer of granulations, occupies its place. This change is produced by an inflammatory process, which gives rise to suppuration and granulation: the bony tissue, beginning with the Haversian canals, is dissolved by the matter secreted within them, while the granulations which shoot forth at the same time, fill up the enlarged canals. The immediate result of this process is the formation of a furrow of demarcation which encircles the margin of the dead bone, and is filled with granulations; and so far as the process is completed on that surface also of the living bone which faces the dead, so far is the sequestrum separated. In this process, that is, in the solution of the layer of healthy bone adjoining the dead by means of the purulent matter, and in the attendant formation of granulations, I find enough to account for the demarcation and separation of the sequestrum; and the absorption which has been assumed to go on at the borders of a portion of necrosed bone I hold to be incompatible with the inflammatory process, while the analogy of the process by which mortified soft parts are cast off renders such a view inadmissible.

The granulations not unfrequently perforate the sequestrum where parts of it are thinner than the rest; and if this should occur at several spots, the dead bone may be completely covered by them. They have the appearance of being developed from the bone, and they fix it so as to delay its removal. It cannot be admitted, even in this case, that the sequestrum undergoes any absorption, but its perforation may be effected by the solving or corrosive power exerted on its tissue by the matter; and this further fact may be attributed to the same power, that, independently of those irregularities on the sequestrum which arise from the unequal thickness of the bone that has perished, that side of it which faces the suppurating tissue appears rough, worm-eaten, discolored, and black. There can be little doubt, indeed, that a sequestrum might be removed in this manner altogether; although at present we are without any observations on the subject made with sufficient care and accuracy to establish it as a fact.

While this process is going on, the dead bone is being replaced by a process of regeneration, which I proceed to describe as it takes place in the different forms of partial necrosis.

In *superficial* necrosis, the inflammation that takes place in the bone around leads to an exudation, which afterwards ossifies upon its surface under the periosteum; and as the inflammation extends more deeply, reaching through the whole thickness of the bone to the inner surface of the medullary canal, and to the spongy diploetic tissue, bone is deposited in those parts also. At the same time, pus and granulations are produced beneath the dead lamella, and the latter form a basis for a new layer of bone. The peripheral exudations first mentioned very frequently grow to considerable dimensions. The earlier the sequestrum is completely removed, so much the sooner does the suppuration cease, and with it the formation of granulations. The latter unite with the cicatrix of the soft parts, and as only a thin layer of them ossifies within the cavity, the scar is generally depressed, and is rendered still more so by the heaping up of exudation upon the healthy bone around.

The muscles remain connected with the old bone for some time after it is dead, reaching it through the openings which are left in the new: but they gradually separate from it, and become implanted in the newly-formed bone (Meckel).

In *internal* necrosis, new osseous matter, generally in considerable quantity, is furnished, chiefly by the outer surface of the bone, and deposited beneath the periosteum, while that membrane becomes closely adherent to the inflamed soft parts around. In the interior of the bone, the separation of the dead piece from the living proceeds in the manner already described, and at length the sequestrum is found enclosed in a shell which consists of a layer of the old bone and a stratum of newly formed osseous substance, and is lined by granulations. This is the sequestral capsule. If this form of necrosis, occurring in a long bone, should involve only a portion of its shaft, the rest of the medullary canal becomes filled up with new bone: should it extend the whole length of the shaft, the exudation then occupies the adjoining spongy tissue of the epiphysis.

The sequestral capsule, so long as the sequestrum remains in it, is perforated by openings, which vary in number, size, and form, and are named *cloacæ* by Weidmann, and by Troja, *foramina grandia.*

Their usual form is round or oval; in size they equal a pea or a bean; and they lead into the cavity of the sequestral capsule directly, or somewhat obliquely through a short funnel-shaped canal: the inner opening of the cloaca is the narrower, and the outer, which is the wider and the more dependent, runs out, in well-marked cases, into a low rim, the lips of which are rounded off. Besides these, there are sometimes other irregular gaps in the new bone, which may be small or very extensive, and the margins of which are sinuous. Most of these openings communicate with abscesses in the soft parts which open externally, whilst others are continuous immediately with fistulous canals that lead outwards through the soft parts.

The openings last named are situated at parts where the necrosis, as it extended from within outwards, has advanced to the outermost lamella of the bone, and where consequently no new bone has been deposited; whilst those first described are formed in connection with the suppuration, and they serve for the discharge of the matter, and for the escape of the sequestrum.

All these openings are lined with granulations which secrete pus, and are continuous with the inner membrane of the sequestral capsule.

As soon as the sequestrum is removed from its shell, the granulations rise from the inner surface of the cavity and fill it, and the secretion of matter ceases. This mass then gradually ossifies, and instead of a medullary tube there is produced a solid cylinder of bone. At a later period, a gradual enlargement which takes place in the Haversian canals of the new bone, changes its structure into cellular, and incompletely supplies the place of a medullary tube.

Thus, when the process of regeneration is completed, the bone consists, from without inwards, of the bone exuded beneath the periosteum, of a layer of the old bone, and lastly, of the central mass of bone which has been produced in the granulations, and which fills up the medullary tube.

At first it has a coarse exterior, is misshapen, thick, and uneven, and a marked boundary is clearly distinguishable between its outermost layer and the surface of the old bone: but as the outer layer becomes more dense and homogeneous with the old bone, this boundary line gradually disappears, the whole cylinder gradually loses its misshapen, thick, and coarse look, and acquires the natural form. This, however, is far from being invariably the case, for sometimes at irregular spots adjacent or superjacent to one another, the tissue of the outer new lamella, as well as of the old compact bone, becomes loose and expanded, and its Haversian canals widened: sometimes its actual mass is increased by internal deposition (hyperostosis interna), the bone retains its coarse appearance externally, becomes very dense in its texture, and is uncommonly heavy. It frequently happens, that the two states are found together, and the latter appears as if it had been developed from the former. In J. Müller's opinion, the so-called new bone, which is produced after internal necrosis, proceeds for the most part from the old bone, the outer layer of which becomes swollen, and grows uninterruptedly during the continuance of the suppuration by the dilatation of its Haversian canals and the formation of new bone in their interstices. In any case, this increase of bulk or swelling of the old bone is to be distinguished from that expansion and swelling upon which Scarpa grounds his theory of regeneration (Miescher).

The mode in which regeneration takes place, when the whole thickness of a bone is dead, corresponds with what has been already mentioned. An osseous exudation takes place upon the outer surface, as well as in the medullary canal of the healthy bone; and after the sequestrum has been removed, granulations continue to rise from the surfaces of the wound, and new bone is formed in them. The regeneration is in most cases incomplete; the growths from the two ends unite too soon, and the bone is diminished in length, and frequently a false joint is formed.

Necrosis of a whole bone is an extremely rare occurrence.

From what has been said, it appears that the regeneration is accomplished by that part of the old bone which has remained healthy; but there can be no question that the periosteum and other surrounding soft parts, and even newly-formed vascular tissue, are capable of furnishing an exudation which will become bone. This is clearly proved by the few cases in which spontaneous necrosis of a whole bone has occurred; by the fact that reproduction ensues in animals when a bone has been entirely removed (Heine); by the appearance of bony substance in the middle of openings made in the skull by necrosis, or by injury, as well as by the occurrence of growths of bone in periosteum and especially in the dura mater.

The foregoing remarks have been directed especially to the long bones, but they apply to necrosis and the process of regeneration in other bones also: only it must be observed, that there is very seldom complete regeneration of any part of a cranial bone lost by necrosis; and when it does occur, it always takes place very slowly (p. 125).

The suppurative process, which ensues upon the death of a bone, may, under various unfavorable local and general conditions, degenerate into an inflammation, attended with an ichorous product, and into caries.

A form of necrosis, differing from that to which the foregoing observations have been especially applicable, is a gangrenous ulceration of spongy bones corresponding to humid gangrene in the soft parts: it is met with chiefly in hospital gangrene, in bed sloughs, &c. The bone is soft and brittle, is filled with dirty greenish shreds of soft parts in a state of slough, and is saturated with a similar sanious fluid.

5. *Expansion, softening, of the tissue of bone, and the consequent indurations.*—Expansion, or rarefaction, though often combined with softening of bone, must yet be distinguished from it. The former is produced by dilatation of the Haversian canals and cells, and constitutes the disease which is named osteoporosis; whilst in the latter there is a deficiency of the mineral constituents of the bone, and some disease of its fundamental cartilage.

Osteoporosis consists, then, as has been said, in an enlargement of the Haversian canals and cells of the bone.

a. This state may result from excessive development of the medulla of the bone, or of the tissues which occupy its canals and cells; while, at the same time, the actual quantity of bony substance remains unaltered. By a rarefaction of its tissue of this kind the bone becomes increased in volume,—expanded. The walls of the enlarging cavities become thinner and thinner, till at length apertures are formed in the interior of the bone, as well as in its outermost lamella, and the cavities communicate with one another. The expanded bone is soft, coarsely porous, and spongy, and more or less so in proportion to the degree of the disease; it yields to the pressure of the finger, and may be easily cut with a knife: its cavities are filled with a large quantity of darkish-red or reddish-brown medulla, which is traversed by dilated vessels, and contains here and there loose or firm clots of extravasated blood.

Osteoporosis may affect the outer compact portion of a bone, and then, in a cylindrical bone, the dilatation of the longitudinal canals gives it the appearance of being split into filaments; or the disease may be developed in the interior in the medullary cavity of a long bone. In the latter case, as the rarefaction advances gradually towards the outer layers of the compact wall, the peripheral lamellar system, though preserving its compact state, becomes distended and bulges all round, the cavity exceeds its natural size, and the bone loses its proper form. The bone is swollen out into a rounded, hollow-sounding, thin-walled cylinder, which is filled with marrow: at its inner wall are found irregularly dilated longitudinal canals, while nothing remains of the spongy tissue and the network in its interior, but a few delicate lamellæ and threads of bone, which pass across the plug of marrow that fills the canal; so that after maceration, the medullary canal is a mere dilated cavity. Of course, the bone is uncommonly fragile, as it is in the allied disease, excentric atrophy.

Lastly, osteoporosis sometimes affects a bone in its whole thickness; and then the disease may have commenced in its interior in the medullary cavity or diploetic substance, or at the exterior, or at all of these points at once.

This kind of rarefaction usually affects the whole of a single bone, or single portions of the skeleton which are intimately associated together, such as the bones of a limb or of the skull; and it affects such bone or bones throughout; more rarely it is partial, i. e. confined to one spot on a bone; the most unusual instance of all is to find it in several bones together. When it is partial it gives rise to the spongy exostosis.

It is most frequently observed in the skull, and advances in that part to a very great degree, the cranial bones in the adult reaching, and sometimes exceeding, a thickness of six or ten lines. It may occur at any period of life, but it is found chiefly in childhood or old age.

This form of osteoporosis, as has been pointed out, is a consequence of excessive development of the medulla of the bone, and of the soft parts which fill its cavities. No distinct general constitutional disease (dyscrasia), can be assigned as its cause, though it is very important to observe, that osteoporosis, whilst it is one way in which rickets exhibit itself, also frequently recurs in old age, in persons who have other marks of rickets upon them.

β. Osteoporosis sometimes arises from an inflammation of the bone and medulla, which furnishes a product in the cavities of the bone, differing in its nature from the ordinary ossific exudation (p. 129). This may be inferred from the traces of recent bony exudation, which are found on bones affected with osteoporosis, and from the fact of the bone beneath soft parts which are in a state of inflammation and ulceration, and that in the neighborhood of caries, being similarly rarefied (p. 133). Moreover, that very painful disease, the malum coxæ senile (which, by the way, occurs in other joints also), appears to originate in a process of this kind: I hold it to be an inflammatory process of a gouty character, which gives rise to rarefaction, swelling, and a peculiar deformity of the head of the femur and acetabulum,—an osteoporosis succeeded by induration.

These cases of osteoporosis are curable.

γ. A large class of osteoporosis is occasioned by atrophy of the bone. The enlargement of the Haversian canals and the cells, is, in such cases, the result of attenuation of the bony lamellæ which form their walls. There is no increase in the volume of the bone, but rather a diminution: it shrinks and becomes smaller: the enlarged cavities of the bone are filled with a gelatinous or fatty substance, which is mostly of a dirty red, brown, or chocolate color. The long bones which have a very thick compact wall, are easily fractured; and spongy bones may be broken into by slight pressure with the finger (osteopsathyrosis). The bones have lost more or less of their weight, according to the degree which the disease has reached, and the patients themselves are specifically lighter than water (Saillant).

Senile atrophy of the bones, as it is the most common instance, may serve also as the type of this form of osteoporosis. But it occurs also in youth and manhood, and is then a painful disease, which usually extends over the whole skeleton, and which it is the custom to ascribe to gout, rheumatism, mercurial cachexia, syphiloid disease, and lepra. In the persons we have mentioned, it sometimes proceeds to such an extent, that in spongy bones considerable cavities are formed, which are filled with the diseased marrow above described; and it predisposes to the occurrence of fractures upon the slightest occasions. The part of the skeleton which suffers least in this form of the disease, is the skull. Like mollities ossium, it has proved up to the present time incurable.

b. There are two forms in which *softening* of bone presents itself, namely, rhachitis and mollities ossium. Some rarefaction is always present in both, but the essential part of the disease is a return of the bone towards its original cartilaginous structure; while at the same time it may be altered in its chemical composition or not. Hence the bones are not brittle, but soft and flexible; they become curved and misshapen, and are much more easily bent than broken.

a. Rickets (rhachitis juvenilis in contradistinction to rhachitis adultorum and rhachitis senilis, which are equivalent to mollities ossium) is a disease of early childhood. It is, in most cases, developed first in the lower extremities: after having reached a certain degree in them, it extends to the pelvis; and advancing from thence to the other bones of the trunk, it at last pervades the whole skeleton. Sometimes it is more prominently marked in one portion of the skeleton, while the rest of the bones are but slightly affected; and then a rickety thorax or skull constitutes nearly all the disease. It is combined with preternatural development of the glandular system, with hypertrophy of the white substance of the brain, with deficient involution, or even with hypertrophy of the thymus gland, with hypertrophy of the spleen, spare muscular development, and a pale and flabby condition of the muscular fibre. It is associated with tubercle very rarely, considering that the deformity of the thorax which rickets frequently occasions, brings on conditions suited to the development of that disease.

It interferes with the growth of the bones in length, and with the development of certain portions of the skeleton in their proper relations as to capacity. Some of the deformities which it occasions are produced only in this way, such as shortness of the long bones, and narrowness and small size of the pelvis; whilst sometimes there are other conditions which essentially co-operate in effecting them. Thus the weight of the body pressing perpendicularly on the pelvis and lower extremity gives rise to the sabre-shaped curvatures of the latter, and the flattening anteriorly, the narrowing of the conjugate diameter, and the great inclination of the former; and this is the case whether the deformity be symmetrical on the two sides, or whether it predominate on one side, and the pelvis be oblique or inclined. Lordosis or scoliosis of the vertebral column follows upon the deformity of the pelvis, and the degree of either is proportioned to that in which the vertebræ are affected with rickets. Consequent upon the deviation of the spinal column from its natural direction, ensues corresponding deformity of the thorax. If the muscles of the thorax—the pectorales and serrati—be in a very undeveloped state, a deformity results which is known by the name of the (rickety) pigeon's breast. In the skull, the hypertrophy of the cerebrum, especially of its anterior lobes, moulds the bone into the peeculiar corresponding shape. The necessary description of all these changes will be given hereafter; only it must be remarked that, as the deformities which are produced by rickets in the lower extremities and the trunk, depend upon causes that vary much in the degree, the duration, and the manner of their action, so they do not follow constantly any definite type, but rather present, especially in the pelvis, frequent exceptions to any forms which may be set down as the rule.

The bones appear swollen out; the angular shaft of the long bones becomes round and cylindrical; and their articular extremities, as well as other broad bones which contain much diploetic tissue (such, for instance, as the bones of the pelvis), become unusually thick.

The texture of the bones is affected in two ways, of which sometimes one preponderates, sometimes the other. In the *first* case the bone is rarefied and increased in size—expanded in fact. A pale yellowish-red jelly is effused into its enlarged canals and cells, into the medullary cavities, and even under the periosteum. The bone itself is abundantly supplied with vessels and full of blood, and its color is therefore darker than natural, and red. Occasionally this change reaches such a degree that the cells of spongy bones, and those in the interior of medullary tubes, become excessively distended, and, as their walls disappear, are merged in larger cavities: medullary cavities at last become single spacious chambers, and the bones uncommonly soft and fragile (Guérin's Consomption Rachitique). In the *second* case the bone is, in addition, deprived of more or less of its mineral constituents; and sometimes it is completely reduced to its cartilaginous element, and appears like a bone that has been steeped in acid. The bony corpuscles are empty, and their rays have disappeared, and when this is the case, the lamellar structure is here and there obliterated; at other parts the lamellæ appear as it were, to have fallen asunder, and the corpuscles are seen quite distinctly interposed between them. It is upon this condition that the softness, the flexibility, &c., of rickety bones depends.

These two conditions exist together, as has been remarked, and sometimes one preponderates, sometimes the other; it is, however, remarkable, that in cases of general rickets, the reduction of a bone to its cartilaginous element so preponderates in some bones as to go on, even to completion, without any trace of rarefaction.

The periosteum of rickety bones is palpably more vascular than natural, and tumid; it clings to the bone so closely that a layer of the expanded spongy tissue always comes away in the attempt to strip the membrane off.

Rickets is not a painful disease. It is usually developed in the second year of life, and leaves traces behind it corresponding to the degree it had attained. In small degrees it is capable of cure by the reabsorption of the substance which has been effused into the cavities of the bone, and the subsidence of the swelling of the bone. In more advanced degrees the cure is effected by that substance becoming more and more firm, and at last ossifying. The bone then remains enlarged and becomes uncommonly dense (Guérin's Eburnéation), and the Haversian canals contract, especially on the concave side of the curves. When the disease reaches its highest degree, the rarefaction which it has occasioned and the fragility of the bone are permanent.

β. Mollities ossium (Osteomalacia, Malakosteon, Rhachitismus adultorum, and senilis), is quite a different disease from true rickets, and affects grown persons in the period between early manhood and old age. It occurs chiefly in the bones of the trunk, to one portion of which it so far confines itself as to proceed to a very great degree in that portion, whilst mere traces of it only are found in other bones. When the bones of the skull and of the extremities are affected, they are so always in a very subordinate degree. It is more frequent in the female sex than in the male; and several times it has been met with coming on after childbed. Not unfrequently it is associated with cancer of the internal organs (a fact which reminds us of the old observation as to the brittleness of the bones in cancer). Sometimes it exists when there is a great production of fat, especially in advanced life: and it is often found when there is also fatty degeneration of the muscles: the import of this last combination is not yet understood, whether it is occasioned by insufficient action of the muscle, or has any essential connection with, and is produced by, the general disease. Compared with rickets, and considering how rarely the disease occurs, its advance to a very considerable degree may be said to be frequent.

The deformities which result from mollities ossium are restricted to the trunk, as has been mentioned above. They take place upon the bed to which the patients are confined, and it is this mode of origin that determines the peculiar shape which results from the disease, and which in the pelvis is regarded as characteristic. The two ends of the trunk approach each other by the vertebral column arching backwards; the thorax sinks in, especially at the sides, the ribs becoming curved and bent in various ways; and the pelvis acquires a triangular form, like that of the heart on cards. But these are not the invariable shapes; and the peculiar form of the pelvis is not exclusively a result of mollities ossium, but is met with sometimes in bedridden persons, who are the subjects of rickets in a high degree.

The bones diminish in size, and their texture is rarefied and atrophied; they become saturated with fat, and reduced to their cartilaginous element. In this condition their corpuscles are empty, and when viewed by transmitted light, diaphanous: there are no canaliculi (kalkkanälchen), and the lamellar structure is lost. The bone at the same time undergoes a striking change in its chemical composition, the extract produced by boiling being not only different from chondrin, but also from the animal matter of bone.

Upon this last-mentioned character of mollities ossium very probably depends not only an essential difference between it and rickets, but also its malignancy: it is a very painful disease, and hitherto has never been cured.

Consecutive induration appears to me to be the mode in which one of the described processes of expansion and softening of bone subsides or heals. The previous occurrence of such a process is at once sufficient to distinguish it from other indurations, but it is characterized also by peculiarities in the texture, and no doubt also in the chemical composition of the bone. The anomaly in the texture of the indurated bone is owing to the rarefaction itself, and to the vascularity of the medulla which occupies the enlarged cavities in the bone, and it consists in the arrangement of the elements of the new osseous tissue upon the old bone and around its vessels, in abnormal relative positions. By this anomaly of texture alone, without reference to any change in chemical composition, an explanation is afforded of several varieties in the physical condition of the bone, such as the peculiarity of its fracture, the appearance of its broken surface, and of a thin section, and its color. Varieties of this kind in a bone, which originate in some peculiarity in the relative position and arrangement of its elementary constituents, have their analogues in inorganic nature, in the different physical condition of bodies which in their chemical composition are alike.

The anomaly in chemical composition may consist in the fundamental cartilage of the indurated bone being overfilled with mineral constituents, the usual proportions of which to each other may be either maintained or altered; or in the presence of unusual salts; or further, in some abnormal condition of the fundamental cartilage itself, of its blastema, &c.

Bones affected with consecutive induration, retain the increased size which they had acquired during their previous expansion, and are therefore of course augmented in weight by the induration.

a. Well-marked specimens of induration may be observed succeeding the expansion which has occurred in advanced life; such cases are most frequent in the skull. A series of skulls of this kind is preserved in the museum at Vienna, and shows, in a most instructive manner, the gradual advance of induration in the expanded cranial bones. Externally, and still more on the cut surface, they present a dull white color, and a chalky appearance; and their fractured surface is coarse. A minute examination of a transparent slip of such bone exhibits wide, irregular, *i. e.* angular, and sinuous Haversian canals: the lamellar structure deficient, or only here and there perceptible; and bony corpuscles, which are mostly round, lying in disorder one over another, and crossing or obliterating each other.

b. Similar characters are presented in the malum coxæ senile, by the indurated head of the femur, and the stalactitic, chalky osteophytes which surround it. It is observed, moreover, that this mass of bone acquires a polish like gypsum. Upon minute examination a close lamellar structure is found: the lamellæ are very numerous, but the bony corpuscles, on the whole, are few, though at some spots they are crowded together in dense groups. The osteophytes present a similar close lamellar structure, and their corpuscles are very numerous and thick, and mostly round and quite black.

c. The induration (éburnéation) in which a high degree of rickets terminates is distinguished by the hardness of the bone, by its glass-like brittleness, and the laminated appearance or leaf-like splitting of its fractured surface. When minutely examined, the Haversian canals are found small, and surrounded by large and widely extending systems of lamellæ, but by few bony corpuscles; those which do exist are small, and, which is remarkable, for the most part transparent, and they have but few canaliculi.

Note.—To this chapter, which contains the greatest quantity, and the most important part of the matter, I subjoin the results of the analyses of several bones, which Dr. Ragsky had the goodness to undertake at request.

1. *Osteoporosis of the skull of an old person.*

Specific gravity,		0·909
Cartilage, fat, and vessels,		38·61 organic constituents.
Basal phosphate of lime and phosphate of magnesia,	55·80	61·39 inorganic constituents.
Carbonate of lime, and other salts,	5·59	

2. *Slight induration, consequent upon osteoporosis.*

Specific gravity,		0·854
Cartilage, fat, and vessels,		44·10 organic constituents.
Basal phosphate of lime, with phosphate of magnesia,	48·20	55·90 inorganic constituents.
Carbonate of lime,	7·45	
Salts soluble in water,	0·25	

3. *The same advanced to a higher degree.*

Specific gravity,		1·842
Cartilage, vessels,		42·51 organic constituents.
Basal phosphate of lime, with phosphate of magnesia,	50·29	57·49 inorganic constituents.
Carbonate of lime and salts,	7·20	

4. *The same at its most advanced degree.*

Specific gravity,		1·751
Cartilage, vessels,		38·27 organic constituents.
Basal phosphate of lime, with phosphate of magnesia,	55·52	61·73 inorganic constituents.
Carbonate of lime,	5·95	
Salts soluble in water,	0·26	

5. *A tibia indurated, also probably in consequence of osteoporosis.*

Specific gravity,		1·490
Cartilage, vessels,		38·49 organic constituents.
Basal phosphate of lime, with phosphate of magnesia,	53·21	61·51 inorganic constituents.
Carbonate of lime,	8·30	

6. *The gypsum-like coating of the head of a femur affected with so-called "malum coxæ senile."*

Specific gravity,		0·845
Cartilage, vessels,		33·90 organic constituents.
Basal phosphate of lime, with phosphate of magnesia,	59·10	66·10 inorganic constituents.
Carbonate of lime,	6·57	
Salts soluble in water,	0·43	

Uric acid, which was looked for particularly, was not present.

7. *A dried scapula, softened by rickets, and a humerus.*

Scapula, specific gravity,		0·612
Cartilage, vessels, fat,		81·12 organic constituents.
Basal phosphate of lime, and phosphate of magnesia,	15·60	18·88 inorganic constituents.
Carbonate of lime,	2·66	
Salts soluble in water,	0·62	

The humerus contained 10·54 per cent. of partly fluid, partly crystalline fat.

8. *Portion of a rib from a skeleton affected with mollities ossium,—the piece was too small for complete investigation.*

Specific gravity,		0·721
Cartilage, fat, vessels,		76·20 organic constituents.
Basal phosphate of lime, and phosphate of magnesia,	17·48	23·80 inorganic constituents.
Carbonate of lime and other salts,	6·32	

9. *The rib of a skeleton in which all the bones were attenuated.*

Specific gravity,		1·432
Cartilage and vessels,		39·63 organic constituents.
Basal phosphate of lime, and phosphate of magnesia,	51·87	60·37 inorganic constituents.
Carbonate of lime and salts soluble in water,	8·50	

10. *Syphilitic induration of the skull in a high degree.*

Specific gravity,		1·613
Cartilage, vessels,		36·30 organic constituents.
Basal phosphate of lime, and phosphate of magnesia,	57·20	63·70 inorganic constituents.
Carbonate of lime,	6·50	

11. *Simple benignant induration of the skull of a lunatic.*

Specific gravity,		1·911
Cartilage and vessels,		33·41 organic constituents.
Basal phosphate of lime, traces of fluoride of calcium,	54·10	66·59 inorganic constituents.
Carbonate of lime,	10·45	
Phosphate of magnesia,	1·00	
Salts soluble in water,	1·04	

N.B. Before determining the weight, each bone was sawn into thin slips, dried quickly in an oil-bath, pulverized, and again dried in the oil-bath at 106° Cels.

Tabular view of the properties of the animal matter from the Bones examined.

No. of bone.	Mode of formation and character of the Animal Matter.	EFFECTS OF REAGENTS ON SOLUTION OF THE ANIMAL MATTER.									
		Alcohol.	Acetic acid.	Tincture of galls.	Neutral acetic oxide of lead.	Basal acetic oxide of lead.	Oxy-sulphuret of iron.	Solution of alum.	Chloride of mercury.	Chloride of platinum.	Ferro-cyanuret of potash.
1.	Glue slowly formed. Solution was whitish, turbid, gelatinous.	Thick Precipitate	. . .	Thick P.	. . . .	. . . . .	. . . .	. . .	Moderate P.	Thick P.	
2.	Changed slowly into glue. Solution contained much fat, and was white, turbid, and gelatinous.	Thick P.	. . .	Thick P.	. . . .	. . . . .	. . . .	. . .	Soluble P.	Thick P.	
3.	Changed slowly. Solution was whitish, turbid, gelatinous, and contained fat.	Moderate P.	. . .	Thick P.	. . . .	. . . . .	. . . .	. . .	Moderate P.	Thick P.	
4.	Changed slowly. Solution of a pale yellow color, rather turbid, had little tendency to become gelatinous.	Turbidity	. . .	Thick P.	. . . .	. . . . .	. . . .	. . .	Moderate P.	Thick P.	
5.	Changed readily. Solution brownish, turbid, and slightly gelatinous.	Slight P.	. . .	Thick P.	. . . .	. . . . .	. . . .	. . .	Slight P.	Thick P.	
6.	Changed with much difficulty. Solution brownish-yellow and translucid, had little tendency to become gelatinous.	Moderate P.	. . .	Thick P.	. . . .	. . . . .	. . . .	. . .	Slight P.	Thick P.	
7.	Changed into glue. Solution brownish, translucid, became slightly gelatinous.	Turbidity	. . .	Thick P.	. . . .	. . . . .	. . . .	. . .	. . .	Thick P.	
8.	Changed into glue. Solution was whitish, turbid, and slightly gelatinous.	. . . . .	. . .	Thick P.	. . . .	. . . . .	. . . .	. . .	. . .	Moderate P.	
9.	Dissolved quickly. Solution was gelatinous, and had a yellowish opacity.	Turbidity	. . .	Thick P.	. . . .	. . . . .	. . . .	. . .	Moderate P.	Thick P.	
10.	Glue formed slowly. Solution was whitish, opaque, and gelatinous.	Slight P.	. . .	Thick P.	Thick P., which was not dissolved either in acetic acid or by boiling.		. . . .	. . .	Thick P.	Slight P.	
11.	The cartilage changed slowly. Solution was whitish, turbid, and gelatinous.	Consid'ble turbidity.	. . .	Thick P.	. . . .	. . . . .	. . . .	. . .	Turbidity	Thick P.	

6. *Adventitious Growths.*—These formations are, on the whole, a rare appearance in the bony system; by far the most frequent of them is cancer. Those which originate in some general diseased condition, are usually the expression of a high degree of it, especially when such diseased condition has the character of dyscrasia: it is, however, a fact of great importance, though it has not yet received much notice, that the dyscrasia, which has established itself in a bone, usually remains fixed there for a long time, and spreads, for the most part, only upon some evident cause, such as forcibly effacing its localized character, that is extirpating the local affection. With regard to that disease, which old writers named spina ventosa, and which has, since their time, been represented in such different forms, I think it best to remark at once, that expansion of bone, from the eventual production of which spina ventosa obtained its character as a disease, is a condition common to several of the morbid growths about to be described.

To the actual new formation in bone, I prefix an account of the morbid development of its system of capillary vessels.

a. Teleangiectasis.—It consists in an enlargement of the system of arterial and venous capillary vessels within the bone. It forms rounded soft tumors, which sometimes pulsate, and which attain various, and occasionally very considerable, size. The dilated vessels produce enlargement of the Haversian canals and cells, expansion of the bone, and subsequently by their pressure, absorption of its substance. Generally, also, the vessels become ruptured and hemorrhages ensue; the extravasated blood forms roomy cavities for itself in the cellular tissue that connects the convoluted vessels, and there coagulates in layers, just as in the sac of an aneurism (Breschet). From special examination of the disease itself, as well as from having met with cancer in other parts of the skeletons in which it occurred, I have been led to believe that it originates in cancer of the bone. In the skull, moreover, I have observed a cavernous structure developed from the diploe.

b. Cysts.—My own experience agrees with published observations of these growths, as to the rarity of their occurrence in bone.

α. The simple cyst, containing a serous or synovial fluid, may occur in any bone, but it is chiefly met with in those of the face, the lower jaw being the most subject to it, and next the upper jaw. In size it may equal a hen's egg, or even exceed it (Dupuytren). By its pressure it produces atrophy of the osseous tissue, and expands the compact tables of the bone to a thin-walled bladder, which crackles under the finger like a piece of parchment. When this layer is also consumed, the cyst protrudes through and beyond the bone, and its wall becomes strengthened by the periosteum, &c.

β. Compound cystoid growths are very rarely seen in bones. Some cases, however, recorded by old observers, undoubtedly belong to this class; especially one or two of those which Lobstein has collected from his own experience and that of others, and has described under the name of *osteolyosis.*

γ. Acephalocysts have been observed in bone eight times. Of these eight cases one, which is preserved in the Vienna Museum, presents considerable interest, from the premises which it affords with regard to the cause of the disease. They have been met with in the humerus, the tibia, the ilium, and the diploe of the skull. In most of the cases recorded by foreign observers, the disease had been developed in consequence of injury. The following is an account of our own case.

The patient, a laborer, aged 42, had, in his youth, suffered from swellings of the cervical and axillary glands; and five years before his death from gonorrhœa and chancre, and consequent bubo. Still later, his penis had been amputated on account of malignant (bösartig) ulcerations; and one year before his death the disease, which was afterwards found in his bones, commenced with pains of a tearing and boring character.

When the body was examined, the left ilium was found converted into a fibrous sac as large as a man's fist, which, besides containing numerous splinters of bone, small and large, sticking in the inner wall of the sac, was also filled with echinococcus-bladders (acephalocysts), varying from the size of a millet-seed to that of a nut. Similar sacs, but less in size, were found also in the pubes, ischium, and sacrum, from which bones they projected into the pelvis. Some of the echinococci were free; but others, especially the smaller ones, were situated either singly or in clusters in the dilated pores and cells of the bare and broken-up pieces of the bone. The bottom of the acetabulum was completely destroyed, and the head of the femur projected into an acephalocyst sac, which occupied its place.

c. Abnormal fibrous tissue.—To this class belong,

α. Fibroid tissue, originally deposited as a product of inflammation or exudation, but arrested in its development into bone; fibroid callus.

β. Fibroid tumors. These occur most in spongy bones, in the articular extremities of long bones, the vertebræ, and the phalanges of the fingers, in the bones of the skull, the lower jaw, and the bones of the pelvis. They sometimes reach a very large size, and distend the bone into a bladder, or so break it up, that it is found scattered in separate fragments through the tumor. The fibroid tumor sometimes has a very dense structure; at other times it is looser, soft, and elastic, and then merits particular notice, inasmuch as it may be easily mistaken in the living subject for other softer—chiefly cancerous—growths; especially if it should have attained a large size, and produced inflammation, sloughing, and ulceration of the integuments by the chafing and pressure which it occasion.

d. Enchondroma.—This growth is incomparably more frequent in bone than in any other structure, and presents in the osseous system all those numerous varieties which are incident to it both in its own internal construction, and in the condition of the bone around it.

It is met with chiefly in the bones of the fingers and toes; it occurs also in the ribs and sternum, and has, moreover, been observed in the bones of the skull, the ilium, and the long bones. Its commencement dates mostly from the period of youth, even though it may have first attracted attention by its enlargement at a later period of life: I have, however, seen cases in which there can be no doubt that it had been developed at an advanced period of life. The variety of aggregated enchondroma I have seen combined with an extensive formation of osteophytes.

Like the permanent cartilages, it generally remains for a long time, and even throughout life, in its original condition; sometimes it ossifies, and I have observed this metamorphosis affecting the last-mentioned variety of the disease in a very remarkable manner; it has been already described. Lastly, an entire enchondroma is sometimes involved in inflammation of the surrounding soft parts, and destroyed (wird verjaucht).

e. Osteoid.—There can be no question that several of the new growths which occur in bone, though they differ in their nature, may be included under this title. Passing by mere concretions, I may observe, that fibroid growths in bone ossify as well as those in other structures: but to be more particular, a spherical osseous tumor may be developed by the progressive ossification of a newly-formed cartilaginous basement in an old bone; and it may be distinguished from the normal bony tissue by the difference of its elementary structure. A most remarkable specimen of this kind is preserved in the Vienna Museum. It is the skull of a person of 26 years of age, who died suddenly whilst suffering from Exophthalmos. In the anterior fossa of the base of the skull on the left side there is a tumor, nearly as large as a duck's egg, which appears slightly lobulated on its surface, and is composed of a very dense, dull white, bony structure: a portion of it as large as a walnut projects into the orbit, and forms one process with another portion, of about the size of a hazel-nut, which extends into the zygomatic fossa. This mass of bone springs from the diploe of the frontal bone, forces its compact walls asunder, and perforates them on both sides. There are other tumors near it, similar but smaller in size, which spring from the diploe of the frontal, and greater wing of the sphenoid, bones.

A different osteoid tumor may be developed also from the enchondroma at any period of its existence.

f. Cholesteatoma is rarely seen in bone, and I am aware of but one instance, which is in the Vienna collection. It is that of an encysted cholesteatoma, occupying the mastoid portion of the temporal, and the adjoining occipital bone.

g. Tubercle.—The frequency of tuberculosis in the bony system is unquestioned. The tubercle either assumes the granular form, or, as very frequently occurs, it is a product of inflammation of the bone, and presents the characters of softening tubercle. Tuberculosis affects chiefly cancellous bones, and portions of bone; the bodies of the vertebræ; the spongy articular extremities of the long bones,—especially the lower end of the femur and upper end of the tibia, and the ends of the bones which compose the elbow-joint; the carpal and tarsal, the metacarpal and metatarsal bones, and the phalanges; and the sternum: more rarely the ribs are attacked, and the cranium: while the parts least frequently affected are the shafts of the long bones.

Its seat is sometimes the outer layer of the bone and the periosteum, and sometimes the deeper bony tissue.

Moreover it very commonly occupies several adjoining bones at once, as for instance, the ends of the bones which form a joint, the whole apparatus of the carpal and tarsal joints, the vertebræ, &c. Young persons are especially subject to it in the years of childhood, and at puberty; but it is also frequent in later, and even in advanced life.

a. Tubercle in the state of gray crude separate granulations can be detected only by close examination of the spongy tissue of a bone in the vicinity of a tubercular abscess. It usually occupies the membrane which lines the Haversian canals and the cells. As the granulations accumulate, they form larger masses of tubercle, and partly compress the bony structure, and partly include necrosed fragments of it amongst them. The aggregate morbid growth is sometimes found as a yellow, lardaceous and cheesy mass; much more frequently it is softened, and consists of a cream-like grayish-yellow pulp, or a thinner, flocculent, tubercular pus. It is contained within a more or less complete lardaceo-callous cyst, which is, in fact, the tissue surrounding the softening tubercle, infiltrated with lardaceo-gelatinous material. If, as is frequently the case, the tuberculous disease should occupy the outer part of a bone and the periosteum, the latter, with the cellular and ligamentous tissues upon it, partakes in the formation of this cyst; and if the tuberculosis advance deeply into the bone, it is mostly the only rudiment of the cyst that can be clearly proved to exist. The best opportunity to observe it, is frequently afforded by the vertebræ.

The degree of congestion which gives rise to primary tubercle, may vary in bone as well as in other tissues; for the development of the disease is, in many cases, unnoticed, while in others it is ushered in with very marked symptoms.

The usual metamorphosis of tubercle in bone is *softening;* but it sometimes also becomes *cretaceous.*

(1.) When it softens, a tubercular ulcer is formed in the bone, which corresponds in extent with the quantity of substance the bone may have lost. The loss of substance arises from necrosis of the portion included in the tubercular mass; and it may die either when first involved in the mass of tubercle, or at a later period: but in either case, it is in consequence of its vessels becoming obstructed, or destroyed in the suppuration.

Softening of tubercle at the surface of a bone, produces a superficial breach of its substance, which has the appearance of being unevenly corroded; when a larger and more deeply situated mass softens, the bone is excavated, and a cavern—a tubercular cavern in bone—results. The greater the number of caverns which are found in a macerated bone, or set of bones, the more safely may it be concluded that they originated in the softening of tubercle.

The cavern contains a fluid, which presents the character of tubercular matter, and is mixed up with numerous particles of bone. Sometimes the particles are small, and resemble crumbled mortar; when larger, they are seen distinctly to be necrosed bone; they are usually of a dirty white color, soaked through with tubercular matter, and not so brittle as the sequestrum produced by other processes in a spongy bone.

The various processes which are usually found in the neighborhood of a softening tubercle, occur in bone also.

First, there is a secondary deposition of tubercles, which, as they soften, increase the size of the cavern. The congestion to which the secondary deposition of tubercle is owing, usually advances to the degree of inflammation (reaction), and leads to the formation of a gelatinous granulating product which lines the wall of the cavern, and as the tubercle softens, always breaks down too. Should the tuberculosis have attacked the peripheral layer of a bone, the congestion, vascularity, and product just spoken of, are seen with remarkable distinctness in the periosteum which immediately covers the diseased spot, and may be found also in the adjoining cellular and fibrous tissues. The periosteum is covered and infiltrated with this product, which, gelatinous at first, gradually assumes a lardaceous appearance. The tubercular matter collected under the membrane swells it out like a saccular appendage.

Oftener still, the inflammatory process becomes more intense, and under the influence of a highly advanced state of the general disease, pervades the osseous tissue throughout with a yellow cheesy product, which breaks down at once. Further remarks on this subject will be given below. It occasions a rapid enlargement of the ulcer in the bone, and extensive destruction, not only of the bone in which it originates, but of other tissues into which it may advance.

When the circumstances of the case are more favorable, and the formation of tubercle has ceased, the inflammatory product at the wall of the cavern becomes organized into a fibroid, lardaceo-callous tissue, and that in the bone itself, into bone; and the cavern changes into a thick firm capsule, which becomes surrounded by an indurated (sclerosed) bony tissue. Its contents are then partly reabsorbed, and partly, as the capsule shrivels and diminishes in size, they become inspissated, and form a greasy calcareous pulp, of a grayish-yellow color, or a mortar which incrusts the walls of the capsule, or a chalky concretion: and thus the tubercular disease is cured.

(2.) Under favorable circumstances, tubercle in bone becomes converted into chalk. A chalky concretion is found in the interior of the bone, enclosed within endurated osseous tissue: if the tubercle have been situated on the surface of the bone, the concretion is covered on the outside by thickened periosteum.

β. A form of tuberculosis, common in young persons, is comprised, in great part, of what are known by the general term of *scrofulous inflammations.*

The inflammation furnishes a tuberculous product by which the actually inflamed spot becomes infiltrated. It may be a primary affection of the bone, or may come on around an abscess already formed from the tubercle above described.

Spongy bones affected with this disease are found at first partly of a dark-red color, injected, and extruding a fatty or gelatinous matter from their cells; and partly pale, and having their cells filled with softening tubercular exudation: both bone and periosteum are frequently swollen, and the former is elastic and soft, and yields easily to pressure or the knife. Ulceration presently begins to destroy it, and the fluid discharged is either thin, grayish, or yellowish in color, and mixed with cheesy flakes, and with particles of necrosed bone; or it is colored of a dirty brown by hemorrhagic exudation, or else is highly discolored, and blackish-green, extremely offensive to the smell, and mixed with black fragments of bone, and with particles of the soft parts destroyed by sloughing.

When this process affects a compact bone, a bone of the skull for instance, its cavities appear filled with tubercular exudation; it becomes of a dirty yellowish-white color, and is, in fact, necrosed—a tuberculous sequestrum. Similar tubercular product is exuded between the surface of the bone and the periosteum. If the process involve only a superficial layer, the bony tissue is partly lost amid the softening of the tuberculous product, and is partly thrown off in particles which are sufficiently large to be palpable; and thus an uneven rugged surface of bone is exposed, from which the process extends more deeply; the osseous tissue previously becoming indurated, and the bone increasing in volume and thickness.

The abscesses which are produced by the process just described, advance in various directions from the bone into the soft tissues, which are infiltrated with the gelatino-lardaceous matter; after having given rise to other secondary (congestive) abscesses in those tissues, they open externally at a part which is often very far removed from their original seat. This is noticed particularly in abscesses in and about the vertebræ.

After the contents of the abscess have been evacuated, or perhaps have partly cretified, the tuberculous caries heals, leaving an indurated cicatrix in the bone, which deforms it in proportion to the amount of substance it has lost: the cicatrix has a rugged and nodular, streaked, and radiated or knitted appearance, and adheres to the thickened and callous periosteum.

The inflammation of the bone which is attended with the production of tubercle, and the caries which thence ensues in the spongy articular portions of the long bones, in the carpal and tarsal bones, and in the phalanges, passed among old writers by the name of Pædarthrocacia, while the same affection of the vertebræ is known by the name of "Pott's disease."

h. Sarcoma and cysto-sarcoma occur in bone pretty frequently; they are sometimes situated on its surface, and sometimes developed in its interior. When deeply seated, they usurp the place of the natural bone, and produce atrophy of it by their pressure; or else distend it so that it forms a more or less complete shell. Just as, usually, the tissue of the bone in the neighborhood of the growth acquires increased density (sclerosis), so, when the morbid growth is superficially seated, a development of new bone takes place, which projects into it in processes like thorns and leaves. And when the morbid growth is of the sarcomatous kind, it is not only encased in an osseous shell, but the bone adjoining the shell enlarges sometimes very considerably, and especially in thickness, while knotted cords of new bone are developed in the tumor, and traverse it in different directions.

i. Cancers. Numerous growths of cancerous nature are met with in bone; they are distinguished from one another by their internal structure and external configuration, as well as by the mode in which they destroy the tissue of the bone.

a. The least frequent is areolar cancer. It forms tumors of greater or less dimensions which protrude from the interior of the bone, and sometimes it exhibits in bone, as in other structures, its remarkable character of developing its peripheral follicles into large bladders or cysts.

have met with a case of this kind in the right superior maxillary bone. n the neighborhood of the canine fossa, a white and densely honey-ombed tissue sprang out of the bone, within the small cells of which a rayish jelly was enclosed: internally it filled the cavity of the antrum Iighmorianum, while externally it grew in the form of bladders, which ttained such a size, that at length those at the periphery of the growth 'ould have contained a hen's or goose's egg, and the whole mass was as irge as a man's head.

β. Fibrous cancer appears sometimes in the form of a nodule, of about he size of a walnut or a hen's egg, which is developed mostly in the iedullary canal of the long bones: it displaces the bony tissue, and pro-ucing atrophy of it by pressure, is frequently the cause of one or more pontaneous fractures of the bone, which occur upon the most trifling ccasion. Sometimes it springs from a broader basis on the surface or i the interior of a bone, becomes a tuberculated and uneven, lobulated iass, and often reaches a very large size: it splits the tissue of the bone sunder into filaments and laminæ; and new osseous substance, com-iencing on them at the base of the growth, and developed continuously long the principal fibres in its interior, forms for it a bony skeleton. This ind of cancer is noticed mostly in the bones of the skull and face, and in ie long bones.

γ. Medullary cancer appears in the following forms:

(1.) In one, which is a rare form, the bone is infiltered with a milk-hite sap,—a fluid encephaloid mass. A case which was long since de-cribed by Saillant, and which has been copied by Lobstein into his hapter on Osteopsathyrosis, ranks, as I believe, in this class: and I am ie rather inclined to think so because my own experience furnishes me ith a similar case. Its rarity will excuse me for detailing it here, in-tead of that which Saillant has already published.

A silk weaver, aged 61, had suffered twenty-five years before death om hæmoptysis, and twelve years before from typhus; since then, from epeated attacks of influenza, and as long as he could recollect, from heumatic pains in his limbs. In the last year of his life he was afflicted ith very severe sharp pains in his lower extremities, and transient dema of the feet. The pains at length extended to the trunk, and ffected the thorax more especially. Fever, cough, and dyspnœa came n; diarrhœa supervened, and the patient died in an extreme state of iarasmus.

Examination of the body.—It was emaciated in every part, and pallid. he bones of the trunk, especially the ribs, sternum, and vertebræ, were ofter than natural: the vertebræ could be easily indented, and contained whitish, milky, and thin or somewhat thick, and creamy fluid, com-osed of round elementary cells. It was mostly unmixed, but here and here it was streaked with some dirty brown medulla. Some of it, in he latter condition, was contained in enlarged cells of the bones of the elvis, and of the articular extremities of both femora and tibiæ. The iner surface of the whole vault of the skull was lined with a pale red, irdaceo-medullary (cancerous medullary) adventitious growth, spread ut in a layer of considerable thickness; into one side of it was inwoven a rowth from the vitreous table, of partly reticular, partly filamentous

Besides the *primary* cancerous diseases of which we have hitherto been speaking as affecting bone, this system is subject also to *secondary* cancer. Instances of the secondary affection may be observed in the ribs and sternum when the mamma is occupied by cancerous disease, in the skull when a primary growth is situated in the dura mater or brain, and sometimes in the bones of the pelvis, when the same disease affects the uterus. The bone becomes involved, not by mere pressure, though that may be exerted, but by the advance of the growth into it; the disease is implanted in the tissue of the bone, which degenerates, and suffers a breach of its continuity.

Bone undergoes a peculiar destructive process, when the soft parts covering it are affected with those phagedænic ulcerations which are usually held to be of cancerous nature. They are mostly observed on the bones of the face, and we shall speak of them hereafter, when taking a comparative glance of the diseases of bone.

Cancer of a bone is sometimes the only instance of the disease in the organism; sometimes, and indeed, very often, several bones are affected together. It is moreover frequently combined with the same diseases in various soft tissues, with cancer in the liver, breast, lung, pleura, uterus, &c. The extirpation of large cancerous growths in bone is usually followed by a very rapid and extensive production of cancer in several internal organs.

It sometimes occurs in early life, but is generally more frequent in adults.

§ 8. *Foreign bodies in bones.*—In some cases in which mercury has been medicinally employed, either internally or externally, particles of the metal have been found in bone. Fragments of all kinds of instruments by which bones have been wounded, may be left behind in the wound,—as broken points of knives and swords, and bullets which have been shot into a bone. They give rise to tedious inflammation, suppuration, and necrosis, and are often thus loosened and cast out; but sometimes they remain firmly fixed in the bone during the remainder of the life of the individual, surrounded by indurated tissue.

An attempt to determine the characters of the Constitutional Affections of Bone, particularly the Inflammations and Caries, by reference especially to the appearance of the Bone after Maceration.[1]

It appears to me to be a matter of much interest to determine in what manner the processes of inflammation and suppuration in bone, which arise from constitutional causes, may be distinguished from one another, and how they may at once be recognized by the characters of the preparation alone: inasmuch as the discovery of certain definite types not only makes us better acquainted with the peculiarities of those processes, but may also assist us in distinguishing them on the living, or at least, in clearing up doubtful cases of diseased bone when examined on the dead.

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[1] Oesterreichische Med. Jahrbücher, vol. ix. p. 4.

The distinctive marks of these processes, so far as they are stamped upon the bone, are comprised in change in its texture at the diseased spot, in some alteration of its shape, in the form and boundary of the ulcer of the bone and the necrosis, in the condition of the neighboring osseous tissue,—that is, in the different degrees, or total absence of inflammatory reaction, and the quantity and arrangement of its product, &c.,—and in the character of the cicatrix. Moreover, although it has only a secondary bearing on our present subject, yet some attention must be paid to the relation which subsists between different constitutional processes and different bones and parts of bones, to their apparent or palpable tendency to affect compact or spongy, broad and flat, or long bones.

In order to render the subject practical, it is necessary to compare as much as possible corresponding bones. This, however, can be carried, out only to a partial extent, inasmuch as several of the processes occur very rarely or not at all, in particular bones; and it is, therefore, the more important to bring together marked examples of the several diseases referred to.

Syphilis, as is well known, most commonly attacks the flat cranial bones, then the tibia and clavicle, and sometimes the sternum,—in general, therefore, bones which are but thinly covered with soft parts, and compact osseous tissue. It makes its appearance as a painful inflammation, which is more severe at some spots than at others. Where it is most severe, it gives rise to a swelling of the bone (tophus), and to an exudation into its interior, which ossifies and produces a local condensation and permanent thickening of the bone; sometimes, but rarely, exudation takes place on the surface also, which soon hardens in the same manner, and unites with the bone. If the bone be diseased throughout, it swells into numerous confluent bosses, which correspond to the several seats of more severe inflammation, and it becomes shapeless, thick, coarse, and heavy.

Or after having effected this metamorphosis, it terminates in caries. Ulceration attacks an indurated bone. Sometimes, especially on the cranium, it spreads over a large extent of the outer table of the bone, while at other times it rather commits its ravages deeply, and, in the skull, often perforates the bone.

In the former case, the destruction of the soft parts discloses a large ulcerating surface, covered with a layer of lardaceous jelly, which is softening and becoming purulent, and beneath which the indurated bone appears rough and uneven, and, as it were, gnawed. If the ulcerative process stop, the layer covering the bone becomes organized into a very delicate cicatrix, and the bone recovers, not with a smooth, but with an uneven nodulated surface, which subsequently becomes somewhat eburnated, but never quite loses its rugged character. If the sutures still exist, their sharp indentations become thick and blunted. These changes may be readily detected on the living patient through the integuments and their cicatrix.

A circumscribed ulcer has the circular or sinuous form of syphilitic ulcers, and swollen thick margins, which in an ulcer that has perforated the cranium, may be rounded off abruptly or bevelled from without inwards

An ulcer which has occasioned a superficial loss of bone, heals in the same manner as the analogous process in the soft parts; its cicatrix is depressed in the centre, nodulated, and shining. If the bone be destroyed in its whole thickness, the margin of the opening becomes rounded, swelled, and coarse, and here and there somewhat inverted. Perforation from this cause happens in the cranial and nasal bones, and in the palate.

And lastly, if the inflammation end in necrosis, the syphilitic sequestrum, especially if it include the whole thickness of the bone, presents the same characters of induration, thickening, and uneven, gland-like ruggedness of surface.

The most palpable characters of syphilitic bones are the hypertrophy, and especially the density of their tissue, and the absence of deposition of bone upon their surface in any of the known forms of osteophyte. Under minute examination of a section of a very compact syphilitic skull, numerous Haversian canals were discovered lying far apart, and separate groups of unusually large and black corpuscles, from which a great number of rays diverged. In a section of a piece of syphilitic skull which appeared porous externally, but was, in fact very compact, the Haversian canals were found wide, the corpuscles mostly large, and some of them placed at right angles to the canals: in the neighborhood of particular canals the innermost lamella was transparent, and contained a single row of corpuscles, but it was surrounded by a dark stratum interwoven with very numerous corpuscles, which were thickly set with rays. The lamellar system of some of the larger Haversian canals was uncommonly developed (mächtig).

A bone which has been indurated by the syphilitic process is unquestionably liable, at a later period, to expansion of its texture. It seems, however, to happen but rarely; I have observed it once in the tibia, but nowhere else,—never in the cranial bones.

Scrofulous inflammation of bone, as it is called, that is, inflammation resulting in tubercular product, and scrofulous caries, have been described already (p. 151), with tubercle of bone, and the tuberculous abscess produced by its softening: the chief characters of the macerated bone are all that require notice in this place.

Caries, when it has arisen from inflammation, is surrounded by a superficial deposition of new bone, which in compact structure, like the cranium, assumes the villous form of velvet, and in spongy bones, especially the articular ends of the long bones, becomes a splintered and plated osteophyte. This deposition is the more distinct, as there is no hyperostosis, no induration in the interior of such a bone until the ulceration begins to heal: and then it commences at the base and circumference of the ulceration. But cases of caries frequently run their course without any such production of bone. They are those which occur in persons exhausted by tubercular phthisis of the lungs and intestines; or when caries in the same kind of persons is secondary, when for instance the articular extremities of bones are affected in consequence of inflammation and suppuration of the adjoining synovial membrane; when the ribs and the sternum are eroded by the compression of pulmonary or glandular abscesses: and also when softened tubercle of the dura mater corrodes and perforates the skull, &c.

Superficial caries, like the analogous ulceration in soft parts, leaves behind it a hardened cicatrix of a round or elongated form, pitted and uneven on its surface, and having a corded appearance, as if it had been knit. The bone often continues permanently diminished in size.

Tubercle, when it softens in spongy bones, as in the bodies of the vertebræ, destroys the bone in rounded spots, which are clustered together so as to give it a honeycombed appearance.

The destruction to which the bones of the face and cranium are subject from so-called facial cancer is altogether different from both these processes. Equally unlike them and every other destructive process, it is distinguished by mere negative marks, and may be recognized at the first glance. The surface of the bone and its diploe are successively destroyed by a kind of dissolution or corrosion; nothing is seen in any part but normal bony tissue laid bare; nowhere is there any obvious trace of expansion of the bone, of induration, or of new bony tissue (osteophyte).

The solution in cases of noma (Wasserkrebs, cancrum oris), affects principally the animal part of the bone, and is very similar to that just described; the bone looks as if it had been calcined (Froriep).

But the most difficult point to determine is the changes which are produced in the structure of bone by genuine arthritis. For there are so many anomalies in those affections of the bones which, on the living subject, are attributed to uncomplicated gout, that one is compelled to doubt whether they are all connected with one and the same process. Adventitious growths of various kinds and metamorphoses of apparently syphilitic nature, are ranged together, under this head, with primary indurations, osteoporoses, with consecutive indurations, atrophy, mollities ossium, with different osteophytes, ivory exostosis, &c.

The following changes may, I believe, be looked upon as arising from gout.

There is a metamorphosis in the bony structure of joints, especially in those of the hip-joint, which I agree with some older observers (Portal, Koehler, Austin) in attributing to an arthritic inflammatory process. It is the same as that which the English denominate "malum coxæ senile." It presents the following characters.

a. The cavities of joints (acetabula) become enlarged, and mostly flattened.

β. The head or convex part within the joint, acquires a flattened surface, and an overhanging margin: in the instances of the head of the femur, of the humerus, of the radius, &c., it assumes the form of a mushroom.

γ. The cartilage which covers the bone is removed, and the cancellous tissue to a varying depth underneath it converted into a dense white, chalky mass, which is polished like marble on its articular surface by constant friction.

δ. An exuberant growth of bone takes place around the joint, in the form of a cup-like and warty stalactitic osteophyte; similar masses accumulate outside the joint, which all consist of the same white, chalky substance as the overhanging margin at the head of the bone.

The process by which this change is produced, is a painful one, consisting, without doubt, in an inflammatory rarefaction, swelling and softening of the bone. After furnishing an osseous exudation within the tissue of the bone and all around,—an exudation which may be distinguished by its form and chemical composition,—it terminates in consecutive induration.

It occurs most frequently in the hip-joint, but it is also observed in the shoulder, elbow, and knee, and in the joints of the fingers, and odontoid process. The whole joint becomes misshapen with the excrescences projecting around it.

The disease in the bone is, moreover, sometimes accompanied by similar osseous depositions in the fibrous capsule of the joint, and in neighboring fibrous structures; they assume various forms, like cups or thorns, or are rounded and bossy.

That inflammation seems to me to belong to the same class, which affects long bones, and besides producing induration of their substance, gives rise to a warty and stalactitic osteophyte upon their surface, which renders them rough, like the bark of a tree. And this may be the case also with the osteophyte that grows in cup-shaped, plate-like, thorny or gnarled, processes, in the substance of ligamentous structures near joints, or on the bodies of the vertebræ. They are very often composed of an indurated chalky substance.

Lastly, under circumstances at present unknown, but especially in aged persons, gout produces a painful atrophy, and concomitant brittleness of the bones, rendering them liable to fracture.

Whether rheumatism gives rise to an inflammation that can be distinguished by any definite characters of its products or to any peculiar caries, is not yet ascertained, however positively assertions be made on the point. There is probably no such thing as rheumatic caries. The abscesses upon and within bone, which have been given out as such, I have always recognized as tubercular. Rheumatic inflammation appears generally to attack the periosteum and outermost laminæ of the bone, and to produce induration of its tissue, and a warty plated osteophyte on its surface. It is evidently a change closely allied to that which arises from gouty inflammation.

Appendix.—Anomalies and Diseases of the Medulla.

Although it is highly probable that the medulla is the part in which all pathological plastic processes in bone originate, yet very little is known of its diseases. And upon this deficiency of information it no doubt depends, that, on the one hand, our opportunities of investigating the diseases of the medulla are almost entirely confined to very advanced cases—cases in which the whole bone is involved in the disease,—and hence that on the other hand, whilst studying the changes in the actual bony tissue, we are in the habit of paying less attention to the medulla.

Oftentimes, in consequence of *hypertrophy*, it is augmented in quantity, and its increase occasions dilatation of the Haversian canals, the cancelli, and the medullary cavities, in which it is contained. Such hypertrophy is unquestionably the cause, either by itself or in combination with other processes, of many of the osteoporoses, both those which are circumscribed, and those which extend throughout a bone. While increased in its quantity, it may retain or depart from its normal texture and composition. Thus instead of the jelly which fills the cells of the spongy tissue of some portions of bones, of the diploe of the bones of the skull, for instance, actual medulla is sometimes found, even in the form of compact lobular masses.

Its excessive accumulation leads at length to atrophy of the spongy and reticular bony substance, and to expansion of the compact walls of the bone.

On the other hand, when the bone is affected with concentric *atrophy*, the medulla shrinks as well as the bony tissue, otherwise its place is taken by a gelatinous, fatty, or serous fluid.

In color and consistence it very frequently deviates from its usual condition, and so also in its texture and composition. The former changes are usually mainly dependent upon the latter in relation to their cause.

The *color* is sometimes unusually pale or white, sometimes it is a dark yellow; and it frequently acquires various hues of red, rusty brown, yeast-yellow, or chocolate, from mixture with blood; it is variously discolored when caries is going on in the bone.

In *consistence* it is sometimes too thin, being liquefied by serum, or oleaginous; in other cases it is unusually firm, it resembles suet or adipocere, and may be broken.

In reference to its *texture*, it is liable to congestion and to hemorrhage, by which it may be discolored, and assume a dark-red, chocolate, rusty brown, or yeast-yellow, hue.

The real seat of *inflammation* in bone is the membrane which lines its cavities. The inflammation of this membrane leads to an exudation which sometimes becomes organized into bone, and sometimes is converted into cellular or fibroid tissue, as may be seen after injuries of bone, or, more rarely, in consequence of spontaneous inflammatory processes; *i. e.*, the medullary membrane and its prolongations undergo fibroid thickening: lastly, the products of the inflammation are sometimes purulent or ichorous, and in various ways destroy the structure in which they are deposited, and the bony tissue. The anatomical marks of these processes are self-evident; they may be recognized also by reference to what has been said about inflammation of bone and its consequences.

In dropsy, the place of the fat of the medulla is gradually taken by a thin, gelatinous, and finally serous fluid.

There are some remarkable changes already alluded to, which the marrow undergoes in osteoporosis and mollities ossium, but their exact nature is still unknown.

Finally, the medullary membrane is the structure in which all *adventitious growths* in bone originate. Tubercle and cancer afford easy proofs of this remark, especially many forms of the latter, such as encephaloid infiltration of bone, and the cancerous diseases named *erosion* by Otto, and *osteolyosis* by Lobstein.

CHAPTER II.

ANOMALIES AND DISEASES OF PARTICULAR PARTS OF THE SKELETON, AND OF THE SEVERAL BONES COMPOSING THEM.

SECTION I.—THE SKULL AND ITS SEVERAL PARTS.

§ 1. *Deficiency and Excess of Development.*—In cases of Acephalus, the skull is altogether wanting, or is reduced to a merely rudimentary condition. It is liable, also, to various degrees of defect, in Acrania being without any vault, and in Encephalocele and Anencephalus presenting various, but less degrees of the same anomaly. Defects of other parts of it are noticed when the cranial or facial bones are fissured, when certain portions of the brain are wanting, or symmetrical parts of it are fused together, as happens in Cyclopia, &c. Examples are also met with in which the development of the skull is arrested in a less degree, apertures closed by membrane being found in its bones, or large membranous interspaces between those that form the cranial vault: the fontanelles are large, or unusual ones exist, or certain sutures continue permanently unclosed. The cases of this class mostly owe their origin to a preternaturally large size of the brain—to Hypertrophy or Hydrocephalus.

The number of bones composing the skull is occasionally incomplete; particular bones are wanting altogether, and sometimes their place is supplied by the enlargement of those in the neighborhood; thus the nasal processes of the superior maxillaries may occupy the space which is left by deficiency of the nasal or lachrymal bones.

An excess of development is observed in those cases where more or less of a second head is formed; and premature closure of the sutures and fontanelles constitutes another, but a less degree of the same general condition.

When certain sutures, such as the frontal, do not close, when there are unusual accessory sutures, such, for instance, as a horizontal one through the parietal bones, but especially when Wormian bones exist, the number of the bones of the skull is increased.

The Wormian bones are most common in the lambdoidal suture, and in the squamous; they are less frequent in the coronal and sagittal, and are most rare where the wings of the sphenoid meet the parietal and temporal bones, and the roofs of the orbits. In the lambdoidal and squamous sutures they are not unfrequently very numerous, and even form two or three rows and as many sutures. When they occur in the other sutures they are often only single; and this is true especially of the sutures surrounding the wings of the sphenoid bone. In their situation, as well as in their dimensions and form, they are usually symmetrical, though there are some interesting cases in which those of the one side do not correspond with those on the other. A Wormian bone situated over a fontanelle receives the special denomination of a fontanellar bone. Finally, the outer layer of the Wormian bones is usually broader than the inner: sometimes they form part of the outer table of the skull only, and, in rare instances, only of the inner. Their existence is chiefly to be accounted for by the large interspaces which are left between the cranial bones in congenital hypertrophy of the brain and in hydrocephalus.

§ 2. *Anomalies in the Size of the Skull.*—The skull, like the brain, may deviate in either direction from its proper size. In some cases it does not reach, in others it exceeds, its natural dimensions. Smallness of size may be general over the whole skull, or may be confined to some particular portion of it. Except when it is occasioned by protrusions of the brain beyond the bounds of the skull (encephalocele), it necessarily involves that the brain generally be small, or that portion of it be deficient or undeveloped; and in the latter case, the corresponding part of the skull is also wanting or but partially developed. The skull may be only relatively small, or it may be absolutely so: in the latter case the smallness of size occasions idiocy, and is a congenital state; a partial diminution is sometimes acquired subsequently to birth, particular parts of the skull becoming small and flattened, sinking in, and shrinking, when the corresponding portions of the brain are in a state of atrophy.

The bones of the face are of small size in cases of congenital hydrocephalus, and their smallness is more striking in proportion to the enlargement of the cranium. A diminution in the size of the bones of the face is also observed in old age: it is chiefly due to wasting of the maxillary apparatus. And a similar attenuation is observed on one side of the face as a consequence of paralysis or neuralgia.

Increase of the size of the skull, when congenital, involves an excessive development of the brain, or, what is more frequent, hydrocephalus. The enlargement is mostly uniform and symmetrical, but in some exceptional cases the skull bulges in one direction or another, a particular section of it is more capacious than the rest, &c.

The dimensions of the skull rarely enlarge at any period after birth —that is, in the sense of increase of its capacity—without some appearance of absorption of the vitreous table, or separation of the sutures; still more rarely does it occur at mature age when the bones are completely formed, and almost never when the sutures are closed. It is seen occasionally at certain parts of the skull.

The bones of the skull and face are subject to many considerable variations in their thickness, sometimes being enlarged (hyperostosis), and sometimes attenuated. Hypertrophy usually commences with the bones of the cranium, it occurs in them frequently, and advances to a very considerable extent; whilst atrophy, especially that form which is peculiar to old age, is more common in the facial bones.

Hyperostosis almost always presents itself in both its forms, namely, that of deposition externally upon the bone, and simultaneous condensation of its tissue (sclerosis): in a few cases it goes on to such an extent, that the skull is not only, according to Jadelod and Ilg, larger than natural, misshapen, and uncommonly thick (9 lines to 1½ or 2 inches), but it also acquires a weight that is almost incredible. In the later periods of the disease, if not at its commencement, the thickening takes place at the expense of the cranial and adjoining cavities,—orbits, nares, labyrinths, and antra Highmoriana,—as well as of the foramina and fissures which are traversed by the nerves and vessels: the sutures also disappear. Other bones, and even the whole skeleton, may be increased in bulk, when the skull is thus affected; but the disease may be entirely confined to the skull, or even to the cranial bones alone, and those of the face, of other parts of the body, and the base of the cranium itself, may remain of their natural size. Not unfrequently, indeed, this hyperostosis of the cranium is associated with atrophy of the bones of the face and of the rest of the skeleton. In any case, it reaches its greatest extent in the cranial vault, and at its frontal and occipital portions: when it occurs in the bones of the face, it is most developed in the lower and upper maxillary bones. It may occur in early youth, in adult, or in advanced life,—a circumstance which depends partly upon the nature of the process.

a. It is sometimes the consequence of an overgrowth or excessive nutrition of the bone, the conditions of which are as yet unknown to us. It is generally developed slowly. It occurs chiefly in adult life; and frequently is associated with ivory-like exostosis on the outer table of the skull, with enlargement and prominence of the inner table, especially near the frontal ridge, and with a growth of bone upon the dura mater.

β. At other times it arises from an inflammation of the bone, which may be acute and recur from time to time, or may be chronic and continued. The pericranium on the one side, and the dura mater on the other, take part in the inflammatory process.

The first of these forms furnishes an exudation of bone upon the surface of the cranial bones, which varies in thickness, and presents the characters of the velvety, finely filamentous, and reticulated osteophyte: it gradually becomes identified with the bone, either with or without the intervention of a newly formed layer of diploe.

Processes of this kind mostly take place on the inner table of the skull, and especially upon and near those spots which are best supplied with vessels; they are, therefore, common along the sinuses and the sutural margins of the bones, and furnish the bone at those parts with a new vitreous table. Moreover, they are processes which occur chiefly in young persons. The exudation of bone, which is met with on the inner table of the skull in pregnant women, deserves an especial notice. It is so frequently observed in women under such circumstances, and advances in them to so great an extent, compared with what it reaches in other cases, that some connection between it and pregnancy must be admitted; and as it has been regarded with interest, since the time of its discovery in this Institution,[1] I devote the following paragraphs to an account of it.

The *puerperal osteophyte*, as it is usually termed amongst us, because we commonly observe it in persons who have died in consequence of the puerperal state, generally occupies the frontal and parietal bones: sometimes it is found covering the whole inner surface of the cranial vault, and in that case it may be noticed scattered in patches over the base of the skull also. But it does not usually occupy large extents of surface completely, even when it is of considerable thickness, the eminences on the inner surface of the skull, and more rarely the depressions, being left uncovered. Such bare spots on the vitreous table, whether they be situated on the eminences or depressions, are parts at which their own pressure, or that of the brain, has prevented the deposition of the exudation, and they are at once distinguished by having lost their polish and natural color.

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[1] Oestr. Med. Jahrbüch., vol. xv. p. 4.

The layer of new bone varies in thickness, from that of a very thin film to half a line, a line, or more. It is usually thickest along the sutures, the longitudinal furrow, and the grooves for the arteria meningea media; it always becomes thinner towards its margins, and is lost in a delicate film.

Its color presents various shades and modifications of red; it almost always becomes paler towards its margin, and at that part is reddish white, white with red beneath, shining through it, or quite white. This depends on the age of the exudation, on the progress which it has made in its change into cartilage and bone, and on the development of a diploetic tissue within it.

It exhibits in its texture the same stages of development as any other ossific exudation.

(1.) It is at first a whitish-red or yellowish-red, gelatinous exudation, which is becoming vascular; it can be easily removed from the bone, and the vitreous table beneath is found to be natural, or to have merely lost some of its polished appearance.

(2.) It is a soft, flexible, cartilaginous lamina, full of minute pores; the vitreous table beneath is generally distinctly rough, or at any rate has its pores manifestly enlarged.

(3.) At the commencement of this stage it forms a flexible lamina, which is smooth and very finely porous, where it is opposed to the dura mater, while on the side which joins the vitreous table it is rough, cellular, and partly cartilaginous, partly osseous. A sanguineo-serous fluid oozes, under pressure, out of its numberless minute pores; and the cellular spaces on the opposite side are filled with a yellowish-red jelly, and sometimes with a clear-red bloody fluid. It is firmly adherent, and is seen, when an attempt is made to remove it, to be united to the vitreous table by the numerous lamellæ and meshes of the cancellous, succulent tissue before mentioned; both these and numerous vessels are torn in separating the osteophyte.

The new growth is not developed beyond this point during pregnancy, or during any morbid puerperal condition which may succeed parturition; but at a later period it ossifies completely, and forms an integral part of the wall of the skull; it becomes, in fact, a new vitreous table, in some instances being dense (sclerosed) all through, in others united to the old vitreous table by an intervening cancellous layer. Generally, when the calvarium is removed, the new growth clings to the inner surface of the skull; but sometimes it separates from the skull, and remains adherent to the dura mater.

When the exudation is more than usually thick and extensive, a similar, but thinner, stratum is found on the outer table of the skull: at this part, also, as on the inner table, it appears to select the frontal and parietal bones, and is deposited chiefly along the coronal and sagittal sutures, and along the part at which the temporal muscle is attached, and the linea semicircularis; it may even be found on the external surface of several of the bones of the face, especially on the superior maxillary and nasal.

That there is no connection whatever between this new growth and the puerperal diseases of which the patients died, will be perceived from the following observations.

It presents itself in all its varieties of extent, thickness, and internal development, in the most rapid instances of puerperal disease: it is met with in cases of speedy death from rupture of the uterus during parturition, and when hemorrhages from that organ, during or after labor, have quickly exhausted the patient, as well as when Asiatic cholera, in its swiftest course, has carried off a woman during her pregnancy. The fact is more clearly proved from these growths being found in persons who, either during their confinement, or soon after it, have died of a disease quite independent of the generative organs, and one which may have arisen a long or a short time before the end of pregnancy, or even during labor, such, for instance, as pneumonia, phthisis, cholera, or apoplexy. Again, there are other cases still more convincing, in which the growth is found in healthy pregnant persons, who have met with unexpected and sudden death at an advanced period of pregnancy. But the fullest conviction is afforded by the discovery of this growth in females, who, at any period of their pregnancy, back to the third month, have died in a rapid or sudden manner.

The osseous growth under consideration, therefore, in pregnant and parturient women, is a phenomenon which, under circumstances hitherto unknown, is attendent on, and originates from, the pregnant condition.

The question as to the period of pregnancy at which the growth commences, is answered by the fact of its having been met with in every month as far back as the third: on one occasion, in which pregnancy was over, and the woman had been confined, it was but little developed, and existed only at a few small spots; on other occasions, at early periods of pregnancy, it extended over large tracts of the skull, its thickness was considerable, and its texture well developed. Its commencement, therefore, cannot be fixed at any definite period of pregnancy.

The exudation when completely ossified, and united with the old vitreous table, increases the mass of the skull in a degree commensurate with its own thickness; and this, of course, is very evident in cases of repeated pregnancy, in which several exudations have been deposited. And a highly instructive fact may be noticed in cases of this class, that some one of the more recent laminæ of bone does not lie in immediate contact with the older layer, but is connected with it by an intervening stratum of diploetic tissue. In those cases in which the dura mater shares in the process, that portion of exudation which it supplies becomes a vascular cellular tissue, and is either spread out as such uniformly, or collected in patches here and there; and the new osseous lamina, being both perforated by the numerous vessels of the cellular tissue and grooved by them as they wind along its surface, does not possess so smooth and polished a surface as the original vitreous table; the dura mater and the skull, therefore, are more intimately connected, and adhere to each other more closely than natural. This connection is most firm, as might be gathered from my earlier remarks, along the sutures and sinuses, and especially near the longitudinal sinus.

What has been said establishes the existence of a puerperal hyperostosis of the skull, and its connection with repeated pregnancies.

In contrast with the frequency with which this growth is found in the bodies of women who either are pregnant or have been recently confined, it is quite rare in other persons, especially in men, to meet with a new formation of bone resembling it in situation, extent, or form. I can recall altogether but eighteen such cases, and the persons in whom it occurred were most of them young, and had died of very various diseases. Exudations indeed are deposited on the vitreous table in both sexes and at all ages; but they are less extensive than the puerperal osteophyte, and are usually confined to the neighborhood of the longitudinal furrow. And very frequently, and even commonly, they are already transformed into a layer of bone, are porous, and covered with serpentine furrows, and have adherent to them a growth of organized cellular tissue, which springs from the dura mater.

The remaining exudations of bone which take place upon the inner surface of the skull resemble needles, splinters, and plates; or they appear as if they had been dropped or poured upon the bone in a fluid state, and had then coagulated.

Chronic inflammation gives rise to a considerable thickening and induration of the walls of the skull, and irregularity and roughness of their surface, with, sometimes, an almost monstrous thickening and fibro-lardaceous condensation of the pericranium on the one side and the dura mater on the other.

This class of disease includes also the hyperostosis of the cranial bones, which originates in their infection with syphilis.

γ. Lastly, the increase of volume, or hyperostosis, may be the induration consecutive upon a rickety state of the bones of the skull, or upon the expansion of bone which resembles that of rickets, but commences at later periods of life. The latter cases are distinguished by the chalky appearance and dull white color of the substance of the cranial bones, and by the coarse grain of their fracture: their surfaces are rough, and the inner table especially exhibits permanently enlarged pores and deep impressions for vessels.

The most extreme cases of hyperostosis are those of chronic inflammation, and the last mentioned consecutive induration. All the rare instances of enormous thickening and induration of the cranial bones appear to be of that nature.

The principal example of partial hyperostosis is the ivory-like exostosis which is frequently observed on the skull. It is almost always combined with considerable induration of the cranial bones. On the inner table, in the neighborhood of the frontal suture, there frequently exist smooth, or rough and striated elevations, which are produced by local expansion and subsequent induration of the skull. Finally, plates or rounded masses of bone, mostly of small size, are sometimes found glued on, as it were, in rugged depressions, on the inner table: they were originally productions from the dura mater.

By far the greatest number of cases of *atrophy* of the skull are those which are peculiar to old age. The bones chiefly affected are the facial, and of these most commonly the maxillary bones. It is an atrophy associated with wasting of the whole skeleton.

A remarkable attenuation of the wall of the skull occurs symmetrically at the top of the parietal bones in old and decrepit persons. At an oval or elliptical spot, the diploe shrinks to such an extent, that the two compact tables unite with one another, and constitute a translucid layer not thicker than a sheet of paper. The diploe around accumulates, so that the bone is thickened externally by an uneven swelling. No internal cause for the appearance has as yet been discovered, though it appears to me not improbable, that it has some connection with inveterate syphilis.

Atrophy is, in some instances, confined to certain portions of the skull; it then presents itself either in the form of diminution of particular parts of the cavity of the skull, or of other cells and cavities in the cranial and facial bones; or it arises, as has been already remarked, from palsy, neuralgia, exhausting reparative processes after injuries, caries, &c.

Absorption of bone (Usura, Detritus) occurs very frequently, and reaches a very advanced degree, in the bones of the skull. In the cranium, when confined to circumscribed spots, it is mostly induced by cancerous growths, such as fungus of the dura mater, or by morbid enlargement of the pituitary gland: when it extends over the whole inner surface of the skull, it arises from hypertrophy, or some displacement of the brain.

§ 3. *Deviations of Form.*—The skull is subject to very various deviations from its healthy form. It will be sufficient to furnish a general account of them, without entering into a detailed description of any but the most important.

Very considerable malformations, both of the cranial and facial portions of the skull, form an essential part of the conditions already described as a hemicephalus, encephalocele, and hydrencephalocele, of congenital hydrocephalus, fissures of the facial bones, cyclopia, &c. In that rare form of encephalocele, in which the protrusion takes place through the ethmoid bone, the vault of the skull sinks down upon its base in the form of a saddle.

In congenital hypertrophy of the brain, but especially in congenital hydrocephalus, the size of the cranium is strikingly disproportioned to the small dimensions of the facial skull. The cranium may be expanded to a circumference of two feet or more; the frontal and parietal bones, especially, being very large, as well as the membranes filling the interspaces between them: the forehead projects greatly, the orbital plates are forced downwards, and so compress the orbits that they become mere narrow transverse fissures: the squamous portions of the temporal bones, and the broad plates of the occipital, incline towards a horizontal direction, the external meatus of the ears are depressed, and the base of the skull, besides being depressed, is remarkably small in proportion to the cavity of the skull. If, despite the large size of the skull, its ossification should be completed, the margins of the bones reach each other by means of long, ray-like denticulations, or they just meet along a sinuous suture (Harmonia), or else ossa triquetra are developed in the interspaces between them. One of the parietal bones, or one half of the frontal, may be increased in size, whilst the other remains unaltered, and then the sutures follow an unusual direction. This is the ordinary form in chronic congenital hydrocephalus; but if, during some interruption of the disease, certain sutures should have closed, a recurrence of the hydrocephalus will produce material alterations in it; the distension will take an unusual direction, and the head deviate, accordingly, from the ordinary hydrocephalic form.

Deficiency in the development of certain parts of the brain produces important deformities of the skull; such as flattening, or receding of the forehead, flattening of the back of the head, &c. The deformity is very striking, when it occurs only on one side. The most frequent deformities are those in which there is a preponderance of some one diameter, so that the skull is longer, broader, higher, or, in some other direction, greater than natural. Allied to these are the round, the blunt, four-cornered, and similar skulls, and those which are oblique. At one time the obliquity is found to consist in a displacement of the halves of the skull in a logitudinal or a vertical direction: and in this way the law of compensation is carried out, the apparently greater width of the cranial cavity on one side being made up for by its condition on the other. When the obliquity is considerable, the facial skull shares in it. At another time, the obliquity is occasioned by a lateral displacement of the several cranial vertebræ (which Carus names Scoliosis of the skull), so that the mesial line of the base of the skull is curved or serpentine. Other obliquities arise from the atrophy of the bones on one side; they are most marked in the face.

Malformations of a peculiar kind, are produced by great projection of the cerebral skull above and in front of the facial; or, as is more frequently the case, by its receding behind the face; the facial angle is consequently either too great or too small.

The base of the skull is sometimes pressed in, in a remarkable manner, by the cervical vertebræ. The skulls in which I have observed this deformity were large, and were those of persons who had suffered, very probably during fœtal life, from a moderate degree of chronic hydrocephalus. The portion which encroached upon the cavity of the skull was, in all cases, very thin. The special conditions under which this malformation occurs are unknown.

Lastly, the skull becomes misshapen in various ways, from fracture, indentation, depression, &c.

The shape of the cavity generally corresponds with the external form of the skull, though it may be altered without any deviation from the natural appearance of the exterior. The inner table is sometimes, as in rickets, unusually smooth and polished; and, sometimes, the elevations and depressions are unusually marked. The elevations and some prominences, such as the clinoid processes, or the eminentia innominata, are occasionally developed into thorn-like, pointed processes, sharp ledges, or shapeless swellings. Sometimes, thorns of this kind are found at unusual places, such as the sella turcica, or on its pommel, on the basilar process, &c.

When deformities of this class occur in cases of hydrocephalus and hypertrophy of the brain, and especially when, at the same time, the subject of them is rickety, the condition of the sutures is peculiar. In a few cases the denticulations form long radiating processes, but generally they are wanting, and the sutures are a mere sinuous apposition of the bones. In hyperostosis of the skull, especially in cases of syphilitic induration, the sutures become changed into a similar "harmonia."

§ 4. *Anomalies in the mutual connection of the Cranial Bones.*—The connection between the bones of the skull may be loosened, and their sutures separated (diastasis). This separation very rarely occurs, and is less important as a result of violent injury to the skull from falls, blows, &c., than when it takes place in consequence of a rapid advance and extreme degree of hypertrophy of the brain, or of hydrocephalus.

The opposite anomaly is that of a premature closure of some, or all, of the sutures. From the thinness of the cartilage the sutures generally unite first at the inner surface of the skull.

Of the bones of the face, the lower jaw may be dislocated, or its joint anchylosed.

§ 5. *Solutions of Continuity.*—The skull is very liable to solutions of continuity, in consequence of its exposure to mechanical injuries. In the infant it may be indented and fissured, or simply indented by the pelvis of the mother, or by the misapplication of instruments to facilitate the birth of the child. Various kinds of punctured and shot wounds, fracture and crushing of the skull, may take place at the spot to which violence has been directly applied; and at the same time bone may be depressed or fragments driven in (impressio et depressio); the tables may be separated and splintered; there may be fissure and contrafissure; the bones may be denuded of their covering of soft parts, or there may be simply concussion of the bone within a circumscribed space. Lastly, the openings made in the skull artificially with the trepan belong to the same class.

All these injuries are of a grave character, not only on account of the violence done to the brain and its membranes by the penetrating instrument or by depressed fragments of bone; but also independently of such complication, from the concussion which the brain and its membranes frequently suffer at the time of injury, as well as on account of the extravasation of blood which takes place immediately beneath the bone, or dura mater, or into the pia mater, or the brain. They may be rendered further serious, irrespectively of the foregoing causes, by the supervention of inflammation of the bone and dura mater, which is usually so much aggravated by the contusion and concussion that have generally happened as to go on to the production of pus and sanies, and readily pass from the external parts to the inner membranes of the brain.

Nevertheless, punctured wounds, fractures, and considerable injuries to the walls of the skull, even when combined with displacement of the fragments, do often heal by first intention, or by the way of suppuration. Fractured surfaces become soldered to each other by bony callus; or, after being rounded off by absorption or exfoliation, they are held together by means of a fibroid callus; and the fragments which perish, and are thrown off during the process of suppuration, are replaced by a similar tissue, in which new bone is sparely formed.

Sometimes one fissure exists alone, sometimes there are several; and, in the latter case, they may all start from one spot on a bone, or may be multiplied by the branching of a single fissure. They often terminate in a suture, which is then usually separated (diastasis), but not unfrequently they are continued across the suture into the adjoining bone. They generally do not unite for a considerable time, and may, even after the expiration of a very long period, exhibit no tendency to union; the rough edges of the fissure are merely rounded off by absorption. If they should then heal, the void is filled up by fibroid callus, which unites with the pericranium and dura mater. They are seldom repaired by means of bony callus, on the contrary, they are sometimes fatal; for the inflammation and suppuration which occur in their vicinity, and in which the inner membranes and the brain become involved, destroy life, it may be after a long period; or the same result ensues, sooner or later, from the various injuries which the brain and its membranes have sustained from the original violence. Large and numerous fissures are generally rapidly fatal, especially such as extend deep into the base, and those which take place from the skull being crushed.

A fall or a blow very frequently produces contusion, separation of the periosteum, or concussion of the bone at a circumscribed spot, while, at the same time, there may be no injury perceptible externally. The consequence is, that the periosteum inflames, pus or sanies is effused, and the outer lamella of the bone may die and exfoliate; or if the bone have been violently injured, it may inflame in its whole thickness, and the periosteum and dura mater with it, and pus or sanies may be produced, both upon the surface and in the substance of those structures: the bone then becomes discolored, and has a dirty grayish-green appearance, all its pores are filled with purulent or sanious matter, its surfaces particularly are rough, and seem corroded, and at length it perishes in its whole thickness. The inflammation of the dura mater very often spreads from its original seat to a considerable extent, and leads to the formation of numerous abscesses, which, after a time, become confluent, and also erode the vitreous table. At length the inflammation spreads to the inner membranes of the brain, or—the pus and sanies being taken up into the veins of the diploe,—either by meningitis or by metastasis, the injury proves fatal.

Openings in the skull, made with the trephine or by necrosis, are closed, as has already been remarked, by means of a fibroid plate, which usually ossifies incompletely or not at all. Complete ossification, when it does take place, always occupies a very long period.

The difficulty with which injuries of the skull are repaired by bony callus is worthy of remark; but the cause of the difficulty still requires a satisfactory explanation.

Fissures are sometimes met with in the cranial bones of new-born children, which extend several lines from the margin into the bone, and usually run a little obliquely through its thickness. It is important to mention them, in a forensic point of view, inasmuch as they bear considerable resemblance to the clefts and fissures which are produced by external violence, and may be confounded with them.

§ 6. *Anomalies in the Texture of the Cranial Bones.*

1. *Hemorrhage.*—Under this head, a disease is included which is of frequent occurrence in the skull of the new-born child, namely, the sanguineous tumor—thrombus neonatorum, cephalhæmatoma,—a disease on which far too much has been written.

It consists of a circumscribed collection of blood, which is poured out beneath the pericranium, in sufficient quantity to form a swelling, that feels doughy or fluctuating. It is distinguished by its situation from the extravasations, which so commonly occur in the new-born child between the pericranium and the aponeurotic expansion above it; and by the quantity of blood extravasated, and the consequent swelling which is formed, it may be distinguished from another extravasation, which forms a thinner layer, with an indistinctly defined margin, on the cranial, and especially on the parietal bones, and is extremely frequent, and indeed almost constant, in new-born children. This last extravasation is indeed of considerable importance, inasmuch as it is only a less degree of that hemorrhage which constitutes the thrombus.

The usual situation of cephalhæmatoma, is the parietal bones, and, as it seems, particularly the right: on this bone, too, it attains its greatest size. It is found with less frequency on the frontal bone, and still more rarely on the occipital. Usually only one exists, and that on one or other of the parietal bones; but cases are not altogether uncommon, in which a second smaller thrombus is found on the frontal bone, and there may be even a third on the occipital. In size it may not exceed a hazelnut, or it may form a tumor extending over the whole parietal bone.

When cephalhæmatoma reaches a considerable size, its shape is remarkable. On the parietal bone, especially, it generally resembles a kidney: its greater and arched margin lies along the sagittal border of the bone, and its concave edge or hylus embraces the parietal prominence. Neither on the parietal, nor on any of the other bones, which have been mentioned, does it probably ever commence on the "punctum ossificationis," but always external to that point. Not unfrequently, however, it spreads over the punctum as well as the rest of the bone.

The cephalhæmatoma is constantly circumscribed near the margin of the affected bone, and does not pass beyond the sutures.

It is a circumstance of considerable importance, though it has hitherto been almost unnoticed, that in very many cases in which there is a collection of blood on the outer surface of the skull, there is also a corresponding extravasation between its inner surface and the dura mater. The inner accumulation is, as a general rule, the less extensive; but there are cases in which the reverse is the fact. Of course, if a bone be laid bare in this manner on both sides for a length of time, the prognosis is unfavorable.

Cephalhæmatoma originally is nothing more than an accumulation of blood beneath the pericranium: there is no essential anomaly either in that membrane or on the bone: most of the blood is usually loosely coagulated, and is of a blackish-red color: and a pale-red fibrinous coagulum, stained with the coloring matter of the blood, frequently adheres to the inner surface of the pericranium, and to the bone.

The examination of a recent thrombus is of itself sufficient to determine as to the truth or error of various statements and opinions that have been put forth, according to which cephalhæmatoma arises from certain anomalies in the development and texture of the bone, and to settle the true nature of the disease and the source of the bleeding. Most of the hypotheses have been based upon examinations made at late periods of the disease, so that following in our description the course of the disease, we shall come in succession upon the various appearances which have been detailed, and which, though correctly observed, have been erroneously interpreted.

When the cephalhæmatoma has existed a short time, appearances are presented precisely similar to those that follow any separation between a bone and its periosteum. An inflammatory process commences, at the margins of the denuded part, and bony matter is deposited in the form of a velvety and finely filamentous osteophyte. The osteophyte extends to a breadth of several lines beyond where the pericranium and the bone remain connected, but it is thickest just at the margin, and there forms an elevation, which rises abruptly around the denuded surface, but externally is gradually bevelled off. This exudation is what has been much spoken of as the bony margin of the cephalhæmatoma, and until quite recently was erroneously regarded as proving some original deficiency, or some loss from disease, of the outer layer of the bone at the base of the cephalhæmatoma. Upon the exposed bone, and inner surface of the pericranium, a fluid next exudes, which is at first gelatinous, but gradually becomes more dense; and it may be observed that the bony margin, just spoken of, becomes continuous with the layer of exudation that adheres to the pericranium, while, at the base of the tumor, it meets that which covers the bone.

Should the extravasated blood be removed by absorption, or evacuated by an artificial opening in the tumor, the pericranium and bone unite together in a simple manner by means of the exudation. But if this do not occur, a very remarkable appearance is presented in a few cases,—few, inasmuch as thenceforward the inflammation usually becomes suppurative. The layers of exudation covering the bone and the pericranium gradually ossify. The denuded surface of the bone and the inner aspect of the pericranium are then each covered with a very delicate and finely reticulated osseous stratum, and the extravasated blood is enclosed between them, and altered to a dirty or rusty brown color. The ossification of both layers of the exudation is sometimes limited to particular spots, and sometimes partial ossification is met with only on that layer which lines the pericranium. In the latter case, plates of bone are found scattered over the surface of the tumor. The sanguineous tumor then exhibits a certain firmness, a kind of rigidity of its walls, and when firmly pressed gives an impression of crepitation like the crackling of parchment. This state of parts may lead to the error, that the outer table has separated from the bone, and adheres to the pericranium.

Far more frequently, when the swelling is not opened and its contents evacuated, the inflammation becomes suppurative, the extravasation changes to a chocolate brown, discolored, fluid pulp, and ulceration or caries, and partial necrosis ensue. The pericranium is attacked with a similar inflammatory process, its inner surface is covered with purulent matter, and the bone becomes rough, unevenly exfoliated, and worm-eaten, and its pores and grooves enlarge.

If the tumor, when in this state, be not opened artificially, or if the pericranium and the other soft parts above it do not ulcerate and make a spontaneous opening in it, the caries which has already commenced at the denuded surface of the bone extends more deeply, an effusion of pus takes place beneath and loosens the dura mater, and at length the bone perishes in its whole thickness. Generally, when this takes place, the pericranium, and the soft integuments covering it, suppurate extensively, and become discolored and easily lacerable. At such a stage as this, cephalhæmatoma generally proves fatal, sometimes by exhausting the strength, but more frequently by the extension of inflammation to the dura mater and inner membranes, and to the brain itself. The fatal result is occasionally brought about by purulent matter being taken into the circulation, and by consequent pyæmia and metastasis.

But even when cephalhæmatoma has reached these advanced stages, a cure is sometimes effected. Healthy suppuration succeeds the evacuation of its contents, and the pericranium unites with the exposed surface of bone through the intervention of a layer of granulations, which afterwards ossifies. The portion of bone which the cephalhæmatoma occupied appears for a long time enlarged, thicker than natural, and somewhat uneven on its external surface, but in process of time this disappears. Even when an extension of suppuration here and there through the bone, and an effusion of purulent matter upon the dura mater, have produced necrosis, and a portion of bone has exfoliated, repair may take place; for granulations arise from the healthy bone which cover the dura mater, and uniting with those that spring from the pericranium, become a basement in which new bone is formed both at the margin of the opening and at other isolated spots.

When there is an effusion of blood upon the dura mater, as well as beneath the pericranium, the exposure of the bone on both sides renders the prognosis of course unfavorable, and the more so in proportion to the extent of the effusions.

Thus, then, cephalhæmatoma consists of an effusion of blood between a cranial bone and its pericranium, and frequently, at the same time, between the bone and its internal covering of dura mater also; and the source of the bleeding is the delicate bloodvessels which pass from those membranes upon and into the bone, and which have been ruptured.

Any essential anomaly in the development of the bone, or morbid affection of its texture, is merely an occasional and exceptional occurrence: the principal anomaly, when any does occur, is that isolated spots of the bone affected with cephalhæmatoma, or of some of the other bones composing the cranial vault, are thinner and softer than natural.

In a recent cephalhæmatoma, however, a manifest congestion of the bones of the skull is pretty constantly observed, and thin strata of extravasated blood may be noticed beneath the pericranium, near it. There is no question that the final rupture of the vessels is due to this congestion; and it is the more certain, from the fact that in ordinary cases there is no other abnormal appearance to which the hemorrhage could be attributed. Moreover, the bone beneath the extravasated blood appears pale, especially when there is an effusion also on the dura mater; and this results from the emptiness of its vessels.

It is an interesting circumstance, that cephalhæmatoma sometimes coexists with effusions of blood between tissues, that have the same relation to each other as bone and periosteum. Thus peripheral apoplexy of a congested liver, or an extravasation of blood beneath its peritoneal investment, is not an unfrequent accompaniment of cephalhæmatoma.

In the great majority of cases, cephalhæmatoma most probably commences during birth, and increases to a palpable tumor soon afterwards. But instances do occur, in which the swelling is not perceptible till several days after birth; and there is nothing against the opinion, that it may form on the skull subsequently to the birth of the child. Its duration may extend over three or four months, or more.

It is most frequently found in first-born children.

2. *Inflammation, caries, and necrosis of the bones of the skull.*—The cranial bones are frequently the seat of these processes, which may be set up not only by violence, but by many other external influences, and very frequently by some internal cause. Sometimes, also, they are occasioned by a previous disease of the bone itself, or by inflammation, suppuration, ulceration, &c., of neighboring parts.

I have stated, in my previous remarks, that inflammation frequently gives rise to an increase of bulk, to hyperostosis of the skull (p. 129). Syphilis, which is a very frequent cause of the enlargement, attacks particularly the frontal and parietal, amongst the bones of the skull, and the nasal bones and alveoli, in the face; and it very generally leads to extensive caries and necrosis. Caries and necrosis, when induced by tuberculous disease, are also observed on the frontal, parietal, and nasal bones; but they are more frequent at the base of the skull, especially in the body of the sphenoid.

As, on the one hand, inflammation may extend from the seat of these processes to the meninges and the brain, so on the other, are they sometimes themselves occasioned by inflammation of the membranous parts of the internal ear, or of the nares and adjoining cavities, or by inflammation of neighboring ligamentous tissues and bones. Thus, for instance, caries of the occipital bone may be caused by inflammation and suppuration of the cervical vertebræ, and of their ligamentous apparatus, and the bones of the face suffer almost incredible devastations from so-called facial cancer.

3. *Expansion, softening, and consecutive induration.*—Rickets not unfrequently, when met with in the infant, exists in a pre-eminent degree in the skull. It may be recognized by the great development of the prominences of the cranial bones, by the small denticulations and sinuous line of the sutures, and by the great thickness, the succulence, the cancellous expansion, the softness, and the great vascular fulness of the bones. It extends to the base of the skull, and, in a small degree,

to the bones of the face also. The swelling of the wall of the skull effaces the inequalities of its inner surface, and it is remarkably smooth: the processes which are situated at the inner surface of the base of the skull, are unusually thick and smooth.

No less uncommon is that form of expansion (osteoporosis) resembling rickets, which affects the skull chiefly, and is indeed generally confined to that part, but which prevails at a later period of life than rickets, and occurs even in advanced age. It reaches, as has been already remarked, a very considerable degree, and terminates in a peculiar and very marked induration of the cranial bones.

When mollities ossium is general throughout the rest of the skeleton, it may affect the skull also; but it always does so in a subordinate degree.

4. *Adventitious growths.*—After the statements which have been made with respect to the occurrence of these growths in the osseous system generally, it is unnecessary, in this place, to do more than to remark, that cancerous growths do frequently form in the skull, as well on the calvarium as at the base: they may all be included, in brief, in the term "*Fungus Cranii.*" They are commonly supposed to be malignant, *i. e.* cancerous, diseases of the cranial bones; but the above-mentioned distinguishing term was given them at a time when an attempt was made to prove the existence and the origin of the same morbid growth in the skull, as had before then been considered peculiar to the dura mater, and had long been named "*Fungus Duræ matris.*" The two diseases may be readily confounded during life, especially when the disease of the bone commences in the diploe; for it breaks through the outer table as it grows, and spreads through an aperture bounded by bone over the surface of the skull.

SECTION II.—OF THE TRUNK AND ITS SEVERAL PARTS.

OF THE VERTEBRAL COLUMN.

§ 1. *Deficiency and Excess of Development.*—Deficiency of the whole vertebral column is met with only in monsters which are very incompletely developed; more commonly, only a portion of it is wanting. The latter deficiency occurs in cases of acephalus, and corresponds in extent with that of the concurrent defect in the neck or trunk. An allied but less deficiency, in which one or more vertebræ or half vertebræ are of small size, or altogether absent, sometimes co-exists with other malformations of the skull and vertebral column, with hemicephalus in the cervical portion of the spine, and with spina bifida: sometimes it is unaccompanied by any anomaly of the kind, occurring in persons who are otherwise naturally formed. In well-formed persons a cervical vertebra is sometimes, but very rarely, wanting; a similar deficiency in the dorsal or lumbar region is less unfrequent. Moreover, the absence of a dorsal vertebra is usually made up for by a supernumerary one in the loins, and the deficiency of a lumbar is supplied by an additional sacral vertebra. It is interesting to observe, in persons who are in no other way deformed, how the want of one half of a vertebra, in the same manner as a half too much, produces congenital lateral curvature.

A faulty and insufficient development of the spinal column may arise from an original fusion of the bodies of two or more vertebræ; this congenital anchylosis is sometimes associated with other malformations, especially with spina bifida and hemicephalus, but sometimes it occurs in persons who are otherwise well formed.

Fission of the vertebral column, spina bifida (Hydrorachis), is an anomaly of great importance belonging to this class. In its nature it resembles hemicephalus, with which it is very frequently combined. It presents several degrees, which are discernible on the skeleton of the vertebral column: sometimes it involves the whole, sometimes only parts of the spine; and its extent, when partial, varies greatly. In its least degree, the half arches are developed, and occupy their natural situation; but, as they have not united, an aperture or fissure remains in the proper seat of union, the length of which depends on the number of vertebræ involved. In a higher degree, the half arches are incompletely developed, more or less of their extremities being deficient, and thus the fissure has a greater transverse diameter than in the preceding degree: usually, also, a larger number of vertebræ is affected. In a condition allied to this, the half arches are fully developed, but stand off from each other to a considerable extent, and are so turned round to the side of the bodies of the vertebræ, that the line of their direction becomes, at length, continuous with the posterior surface of the bodies: they are usually then flattened from before backward; and, as in the form already mentioned, are here and there united with one another. Although there is no actual deficiency of development, the fissure becomes very wide, and diminishes in depth. In a still higher degree, the fissure involves not the half arches alone, but also the bodies of the vertebræ; and, in the highest degree of all, one of the half arches may be wanting, and a part, or even the entire half, of one or of several of the bodies. Fission of the vertebral column in its whole length, or of its cervical portion, scarcely ever occurs, unless hemicephalus and hydrencephalocele exist also. The most common situation of spina bifida is the lower dorsal and lumbar region. Fission of the sacral vertebræ is more rare: sometimes it occurs in two places together, and then usually one fissure is in the neck, and combined with hemicephalus, while there is another in the lumbar or lower dorsal region.

Excess of development is exemplified in the presence of an unusual number of whole or half vertebræ. In the former case, there are sometimes thirteen dorsal, or six lumbar vertebræ; the vertebral column is, to a corresponding extent, longer than natural; and, connected with the supernumerary dorsal vertebra, there is an additional rib. An excess of one or more halves of vertebræ occasions a congenital lateral curvature, in the same manner as a deficiency of halves of vertebræ; and it constitutes a most remarkable instance of scoliosis, of which I shall treat more at large hereafter.

§ 2. *Anomalies in the form of the Vertebral Column, and of its several Parts.*—Deficiencies of development involve, as has been stated, various anomalies in the shape of the several vertebræ, and also, as will further appear, deformities of the whole column. Moreover the approximation to each other in form, which the vertebræ exhibit at the limits of the natural divisions of the column, sometimes gives the appearance of a vertebra being deficient. In this manner the last dorsal assumes much of the character of a lumbar vertebra, and more frequently the last lumbar becomes a sacral bone: this transference from the lumbar to the sacral region may be symmetrical, and occur on both sides, or may take place on one side only. On the contrary the first lumbar may approach a dorsal vertebra in character, and sometimes it bears the rudiment of a thirteenth rib: or the first sacral vertebra may resemble the last lumbar. Finally, the vertebræ are subject to manifold deformities at different periods of life, in consequence of exostosis, osteophyte, and partial absorption of the cicatrization which succeeds the loss of substance occasioned by caries and necrosis, &c.

Some of the deformities of the vertebral column are congenital; others, and those the greater number, come on at different ages after birth, and consist of various forms of curvature of the column. Those of the former class are for the most part occasioned by so serious affections of the central organs of the nervous system (hydrorachis combined with anencephalus, encephalocele, &c.), that they very rarely come under observation at the later periods of life. The deformities produced by high degrees of fission of the vertebral column, and the curvatures which accompany them, are instances of this kind. In other cases the curvature of the spine, and the other deformities coexisting with it, are produced by the contraction of muscles, to which certain diseases of the nervous centres give rise. Sometimes the curvature results from deficiency of the lateral half of a vertebra, or from unequal development of the two halves of the column, or from the presence of one or more half-vertebræ too many. Lastly, fission of the thorax or abdomen, or *eventration*, may make the spinal column deviate from its natural direction. The form of the deviation may vary; it may be a simple curvature, or, as is the case with those which come on after birth, it may be a compound of two or more curves, &c.

I venture to introduce in this place the description of three cases of original deformity of the spinal column: they are of rare occurrence, and the first of them is perhaps unique.[1]

CASE I.—*Compound Scoliosis, occasioned by the presence of supernumerary lateral halves of vertebræ, which compensate each other.*—The spine of a woman, æt. 46, a very old preparation in the Museum at Vienna, but unfortunately not made with care proportioned to its value.

The sacrum and coccyx are united into one bone, on the right side of which there are four sacral foramina, and five on the left; for the first sacral vertebra is higher on the left side than on the right, and, as is evident, from its left spinous and articular processes being double, it consists on that side of two lateral halves of vertebræ fused together. The fifth lumbar is developed on the right side to a sacral vertebra, and thus the height of the left half of the sacrum is level with the right.

The first lumbar vertebra, in the concavity of the lumbar curve on the left side, appears very depressed, being not more than eight lines in height, and is concave from above downwards; while on the right side it is convex, and more than two inches high, and has a horizontal groove filled with an ossified intervertebral body, which indicates that it is double on that side. On the left side there is but one half arch, on the right there are two; there is also a small supernumerary intervertebral foramen on the same side (the right), and a half spinous process which has no fellow.

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[1] Oesterr. Med. Jahrb. vol. xix.

This odd half spinous process alters the position of the bodies of the vertebræ, and more particularly of their spinous processes above and below, in such a manner, that the laminæ, more or less displaced and overlapping, terminate in a row of unsymmetrical spines. Inferiorly, the derangement stops at the second lumbar vertebra; but above, it reaches to the eighth dorsal; the right half spines of the first and second lumbar vertebræ lie beneath those of the left side: but the right half spines of the twelfth, eleventh, tenth, ninth, and eighth dorsal, are placed above those of the left side; hence they either appear unsymmetrical, or here and there one of the right half spines comes in contact with the left one of the vertebra next above.

The left half of the seventh dorsal vertebra, on the convex side of the inferior dorsal curve, is very high; it is pretty distinctly marked with a horizontal fissure in the same way as the first lumbar vertebra, and has two half arches instead of one, just as the first lumbar has on its right side. The lower one, which is the thicker, unites with the single arch that exists on the right side, and both together form a complete spinous process; the upper one terminates in an odd spine. There is an intervertebral foramen between the double arches, which is rather smaller than the foramina adjoining it above and below. The sixth dorsal vertebra has an apparently odd arch on the right side, which is adapted to the supernumerary half spine of the seventh dorsal vertebra, while the left arch, as will presently appear, is shrunken and combined with the corresponding arch of the fifth.

Between the sixth and fifth dorsal vertebræ, on the right side (at the convexity of the upper dorsal curve), another, a fourth, half vertebra is intercalated, which has a half arch on the right side. Its spinous process unites with the combined left half spines of the fifth and sixth dorsal vertebræ.

The fifth dorsal has an arch, the left half of which (at the convexity of the upper dorsal curve) is increased in breadth by union with the left half arch of the sixth dorsal. It has but one transverse process, and it unites with the half arch of the intercalated half vertebra to form one very broad, flat, spinous process; while it forms another, and more slender one, with the corresponding right half arch of the fifth.

The half arches of the fourth dorsal vertebra lie one over the other, the left uniting with the slender spinous process of the fifth (fifth and sixth), whilst the right terminates in a half spine.

The third dorsal is tolerably well formed; but the right half arches of the second and first coalesce, and their single spinous process joins with that of the left half arch of the second, while the other half of the first terminates again in an odd spine.

According to this, therefore, there are in the dorsal, lumbar, and sacral parts of the column, four half vertebræ, with their half arches and processes, too many. They are so placed on the two sides as fully to compensate one another; for upon the duplication of the left half of the first sacral vertebra there follows duplication of the right half of the first lumbar: and then, as the left half of the seventh dorsal is double, there is half a vertebra interposed on the right side between the sixth and fifth dorsal. And with regard to the arches,—the half arches of the sixth and fifth dorsal coalesce on the left side, and those of the second and first dorsal on the right.

Lastly, as has been already pointed out, there result from the position of the abnormal half vertebræ the following curvatures of the whole column:

α. Curvature of the sacrum, with the convexity towards the left, in consequence of duplication of the left half of the first sacral vertebra: the development of the fifth lumbar vertebra to a half right sacral compensates this curve.

β. Slight curvature in the lumbar and lower dorsal regions, in consequence of duplication of the right half of the first lumbar vertebra: the convexity at this part is directed towards the right.

γ. Considerable curvature in the middle dorsal region produced by the left half of the seventh dorsal vertebra being double: here the convexity is towards the left.

δ. Considerable curvature in the upper dorsal region, which is caused by the half vertebra interposed between the sixth and fifth dorsal vertebræ: the convexity here faces the right. The last two form a very compressed S curvature; and the vertebræ are twisted upon their axes, and project backwards (kyphosis).

Corresponding to the anomalies in the vertebral column, there are some very remarkable peculiarities in the number, form, and attachment of the ribs. As there are two supernumerary half vertebræ in the dorsal region, one on the right side, and the other on the left, that is, one supernumerary dorsal vertebra, and the number of articulating surfaces on the bodies and transverse processes being in accordance with that number of vertebræ, there should be thirteen ribs on each side: but there is another attached to the seventh cervical vertebra, and there are actually fourteen more or less complete ribs on each side.

The first rib on the left side is attached by two heads, the upper one of which articulates with the seventh cervical vertebra just above its lower border, and the lower with the first dorsal: the two heads unite in a single neck; the tubercle divides, and is applied to the transverse processes of the seventh cervical and first dorsal vertebræ; and the rib then ends in a single shaft. The first rib on the right side is also attached by two heads: the upper, which is the thicker, and has a cloven neck, joins the seventh cervical vertebra opposite the upper head of its fellow; the lower head is more slender, but sinks deeper into an excavated articulating fossa between the first and second dorsal vertebræ. The three necks soon unite into a single broad one, which is attached by one tubercle to the transverse process of the seventh cervical vertebra, and by two others to a very large articular process on the coalesced right half arches of the first and second dorsal vertebræ; it then separates into two distinct shafts.

The third and fourth, or rather, if we enumerate by the heads of the ribs, the fourth and fifth, ribs on the left side have but one neck, and for a short distance also, only a single body. At the single very thick transverse process of the united left half arches of the fifth and sixth dorsal vertebræ, two ribs are attached, namely the sixth and seventh.

Moreover, anchylosis has taken place between the odd half arches and bones next adjoining them, between several of the bodies of the vertebræ, especially in the concavity of the curvatures, and also between the second and third cervical vertebræ.

CASE II. *Scoliosis produced by deficiency of one half of a Vertebra.*—The spine of a tailor 70 years of age.

It consists of the cervical skeleton (excepting the atlas), of twelve half dorsal vertebræ on the left side, and eleven on the right, of four abdominal and four sacral vertebræ.

The six inferior cervical vertebræ form one curved hump: their bodies and articular processes are united, each to each, into one piece, of coarse cellular structure, the anterior surface of which looks as if the bony material had been poured over it in a fluid state and had then coagulated; while a tense, and partly ossified, ligamentous tissue, stretches down over the arches.

The sixth and twelfth dorsal vertebræ form the extremities of a slight curvature to the left, in the concavity of which (on the right side) there is half a vertebra wanting. For only the left half of the ninth dorsal vertebra exists, which is united to the eighth dorsal, and with it composes one very high body and a similar half arch on the convex side of the curve. There are two transverse processes of nearly equal size upon the half arch, and two spinous processes which lie one above the other, but are fused together.

All the dorsal vertebræ, from the sixth to the twelfth, are connected together, anteriorly, by a mass of bone, partly cellular and partly compact, which is most abundant over the intervertebral bodies, and looks as if it had been poured out upon them. Their articulations, also, are more or less completely anchylosed.

The spinous processes of the last two (the third and fourth), lumbar vertebræ, and the left transverse processes of the second and third, are driven upwards by a deviation of the sacrum considerably backwards and a little to the left; and the third and fourth spines are, at the same time, pressed together. The last lumbar is converted into a sacral vertebra.

The sacrum curves strongly backwards and to the left: it consists of four vertebræ; the last two of which, especially on the right side, in consequence of the displacement of the anterior and posterior sacral foramina from their natural positions, resemble a sieve perforated with large holes.

CASE III. *Angular Curvature (kyphosis), produced by the twelfth dorsal Vertebra consisting of two divided lateral halves.*—The spine of a woman, æt. 55. The two portions form triangular rudiments inserted laterally between the eleventh dorsal and first lumbar vertebræ, with their points directed inwards: and they are united with the first lumbar in such a manner, that its body is very high at the sides, whilst in the middle it seems low, and is in contact with the eleventh dorsal. In consequence of this deficiency in the mesial line, the vertebral column is bent backward at a very obtuse angle. The arch of the divided twelfth dorsal vertebra is completely united into one piece with that of the first lumbar; but the half spines of the latter are so twisted, that the right one appears to be higher than the left. The last right rib is connected, by two heads, with the twelfth dorsal vertebra.

The curvatures which are acquired may be divided, as they are naturally, into the three cardinal forms: of curvature to either side, lateral curvature—*scoliosis;* curvature backwards, or angular curvature, the hump—*kyphosis;* and curvature forwards, sinking of the back—*lordosis.* Scoliosis, as will be seen in the sequel, may be combined with the other two curvatures. Moreover, it is of importance to distinguish the primary deviation from those which are consequent upon it, and compensatory.

a. Scoliosis is by far the most frequent of the three. There is sometimes only one curve: but more commonly a second, a compensatory inclination towards the opposite side at some other portion of the spine, renders the deviation sigmoid: in some cases there are several curves, and in others the scoliosis is combined with obliquity of the pelvis. When the lateral curvature is double or sigmoid, it is usually the dorsal region and the lumbar that are bent; and though the primary curvature may be in either region, it is far more frequently in the dorsal than in the lumbar. And further, the deviation in the dorsal region inclines most frequently towards the right side; whilst the compensatory curvature in the lumbar region to the left is itself compensated by a corresponding obliquity of the pelvis. In order to make even the little that has been said intelligible, as well as the statements which follow, it is necessary to point out the chief causes of scoliosis.

Attention has already been directed to the deviation of the spine which results from original inequality in the lateral halves of the vertebral skeleton. It is that to which Guérin attributes those cases of hereditary scoliosis which become perceptible between the ages of 7 and 10 years, and are generally unaccompanied by any trace of a rickety constitution. There is also a curvature to which the female sex is liable, in which Guérin ascertained that a disproportionate growth or elongation of the vertebral column, takes place at the period of puberty. But lateral curvature of the spine may come on at various periods of life subsequent to birth, under other conditions, which are as follow:

α. Active muscular contraction, arising from some idiopathic and substantive, or from a secondary, affection of the nervous system, especially of the nervous centres, and usually combined with other deformities of the skeleton, contractions, palsies, &c., which also owe their origin to muscular contraction.

β. In most cases it arises from neglecting or impeding the action of the muscles of inspiration of one side. All the scolioses traced by Stromayer to paralysis of the muscles of inspiration on one side are of this class, as well as those in which the spine is bent in consequence of chronic pleurisy, and narrowing of one side of the thorax. In such cases, the primary curvature is at the dorsal part of the column; and as, in most occupations, it is the left side of the thorax, the function of which is impeded or neglected, the spine inclines far more frequently towards the right side than towards the left.

γ. The curvature may be in consequence of rickets: the pelvis deriving some one-sided deformity from the lower extremities, propagates it to the vertebral column. The first curve is then in the lumbar region, and that in the dorsal region is consecutive, and slighter than the other.

δ. Lastly, scoliosis may ensue after a one-sided deformity of the pelvis has been produced by dislocation of either femur upwards, whether the dislocation be the result of violence or of disease (spontaneous).

Every considerable lateral curvature is accompanied by a twisting of the vertebræ upon their axes; and this rotation or torsion is of importance in reference to the diagnosis. The vertebræ are always turned towards the curved side, that is, the bodies face the convexity, and the spinous processes the concavity of the curve: that vertebra is the most rotated which forms the most prominent point of the curve, and for the same reason, the spinous process of the same vertebra is the deepest in the row of distorted spines. Upon these facts Guérin rests the solution of the problem, from a given curvature of spinous processes to determine the degree of deviation of the vertebral bodies.

This rotation of the vertebræ upon their axes produces the deviation of the vertebral column backwards, and the rounded hump, which are the peculiar result of lateral curvature (*excurvation* of Bampfield). It is a compound distortion, which has been named *kyphosis scoliotica;* but which, inasmuch as the lateral curvature—the scoliosis, is the primary evil, would be better called *scoliosis kyphotica.*

The changes which take place in the several vertebræ and their processes, as well as those in the ribs, become more palpable in proportion to the degree and the duration of the lateral curvature. The vertebræ on the side of the concavity are less deep than natural; sometimes they scarcely measure one-third of their normal height; they slope considerably from above downward, their margins seem pressed forward, and their form, in short, bespeaks their having undergone gradual compression. The intervertebral bodies are more or less wasted, and sometimes are entirely removed: when this is the case the vertebræ at last become anchylosed. The processes in the concavity of the curvature become slender; they are frequently elongated, pressed together, and flattened against one another, and the articular processes are anchylosed. And the ribs, in like manner, attenuated, compressed, and flattened where they adjoin the vertebral column, become anchylosed to the vertebræ and to one another.

b. Curvature of the vertebral column *backward* presents itself either as an arching of its dorsal portion, a morbid excess of the natural curve in that region (excurvation of Bampfield), or in the form of the hump,—kyphosis, gibbus (angular curvature, angular projection of Bampfield). The former is a common occurrence in old age, or in consequence of mollities ossium; the latter, which is more the subject at present under consideration, is produced almost always by inflammation and caries, but sometimes by fracture of the bodies of the vertebræ, or by inflammation and suppuration of the intervertebral bodies. With occasional exceptions we may say, with Meckel, that this curvature is more important in proportion as there are fewer vertebræ affected, though the greatest curvatures are those in which whole vertebræ on the bent side are destroyed in considerable numbers, and those which remain are united to one another.

The most common situation of the disease is the lower dorsal and upper lumbar regions, though it does occur also in the upper dorsal, and even in the cervical and the lowest part of the spine.

Not unfrequently the lateral halves of the diseased vertebræ are destroyed unequally: the upper part of the column will then incline to one side.

c. Curvature forward—lordosis—is met with in greatest extent and frequency in the lumbar region: it scarcely ever occurs as a primary curve in that situation, but is almost always consecutive upon some previous one, compensating, as such, the obliquities of the pelvis produced by rickets, congenital lameness, or coxalgia on both sides. It is sometimes not limited to the lumbar region; but occurs, as a consequence of considerable angular curvature, in other parts also. Lastly, it sometimes comes on in the course of diseases of the spinal cord and of palsy, and extends the whole length of the vertebral column. Sometimes, when it is consecutive upon deformities of one side of the pelvis, arising from rickets or coxalgia, a certain amount of lateral curvature is combined with it.

Every primary curvature is compensated by a second curve in the opposite direction, which generally occupies the part of the column immediately adjoining the first; not unfrequently the second is succeeded by a third, and that even by a fourth. Upon this fact depend the various consecutive deformities which the pelvis presents in regard to its form, symmetry, and position. In cases of lateral curvature, not only is the primary deviation in the dorsal region followed by a lumbar curve in the opposite direction, or *vice versâ*, a primary lumbar by a dorsal curve, but the primary curvature reaches along the column in extended sequence, in such a manner, that the second curve is compensated by a third, and this again not unfrequently by a fourth. The following condition is quite common: a primary curvature to the right in the thoracic portion of the spine, is followed by another to the left in the lumbar region, and the rotation of the vertebræ corresponds with the amount of the dorsal curve. But the sacrum exhibits a deviation, which commences, perhaps, at the lowest lumbar vertebra, and takes an opposite direction to that of the lumbar curve; that is to say, the sacrum appears lower than natural on the left side, and higher on the right, and betrays the rotation of its component vertebræ towards the side opposite to the lumbar curve, by projecting into the pelvis on the left side deeper than on the right. Lastly, in many cases the coccyx projects in an opposite direction to the curvature of the sacrum, and forms a fourth deviation of the column.

In another case, a primary deviation of the sacrum of a rickety pelvis to one side and backward (inclination of the pelvis on one side to an unnatural degree), may be counterbalanced by a curvature of the lumbar vertebræ to the opposite side and forward (*lordosis scoliotica*), and this, again, may be equalized by a dorsal curve in the contrary direction.

The amount of the compensating curvature generally equals that of the primary curve: but to this there are frequent exceptions; and the second curve may at one time be quite subordinate to the primary, at another may considerably exceed it.

Kyphosis, or the angular projection, which is formed by two sides of an angle, is counterbalanced by a curvature forward (lordosis). The compensation is sometimes effected chiefly by the upper, sometimes by the lower part of the spine: as a general rule, it is the longer side of the angle by which the balance is restored; though sometimes its two sides are equally curved. These compensatory curves, again, whenever it is possible, become the occasion of further curvatures in the opposite direction; and the cervical vertebræ and sacrum, by their projection backward, make up for the deviation of the adjoining regions forward.

The compensation in *Lordosis*, or curvature forward, is obtained in two ways, according as it arises from too great inclination of the pelvis, or from angular projection in the dorsal region. In the former case, the natural curvature in the dorsal region is slightly increased; in the latter the sacrum, and therefore the pelvis, incline backwards.

When there is a combination of lateral and angular projection, or of lateral, with a primary curvature forwards, the form of the compensatory distortions of course corresponds with those of the compound primary curves.

Distortions of the spine diminish the capacity of the two great cavities of the trunk to such an extent as materially to interfere with the development and free action of the thoracic and abdominal viscera, while they also produce various changes in those organs, as regards both their position and their form; but the most serious consequences are those which ensue from extreme lateral and angular curvatures, as such distortions narrow and deform the thorax, and impair the functions of the lungs, and consequently of the heart: They occasion an increase of density in the tissue of the lung, and thereby give rise to active dilatation of the right side of the heart, and enlargement and permanent congestion of the whole venous system,—cyanosis. And hence, though a contrary opinion is very commonly entertained, they establish that general immunity from tuberculous disease, of which I have already spoken.

The deformities of the thorax and pelvis resulting from curvature of the spine are so intimately associated with the subject before us, that they must be treated of at once; and it will add to the interest with which we shall enter upon the study of the distortions of the pelvis in general, and more especially of the consequent curvatures of the spine, when we can refer again to our present conclusions, and find how the results of analysis in the two cases agree.

1. *Thorax.*—The most extreme deformity presented by the thorax is that which occurs in lateral curvature, and in the combination of lateral with angular projection. It seems displaced in the opposite direction to the convexity of the dorsal curve, and the whole, or more commonly the lower end only, of the sternum, swerves from the mesial line in the same direction; the axis of the thorax itself inclines towards the convex side of the dorsal curve. One consequence of this deviation is, that that half of the thorax which is on the convex side of the curve is lower than the other, and approaches the pelvis; when there is considerable curvature, the false ribs touch the ilium, or even project into the iliac fossa. But

in extreme cases of combined lateral and posterior curvature (scoliosis kyphotica) in the lower dorsal region, the thorax assumes the contrary position; the ribs which pass from the concavity of the curve force the chest to the opposite—the convex side; the sternum diverges in the same direction, and the sunken half of the thorax is that on the concave side of the curve.

Moreover, both sides of the thorax are flattened; and the amount of flattening is proportioned to the extent to which the vertebræ are twisted on their axes, and consequently to the size of the hump: but it is always most marked on that side of the chest which corresponds to the concavity in the spinal curve, and which may even be indented, while the other side is somewhat vaulted. The ribs take a more direct course outwards and forwards in proportion as they emerge from the deeper part of the concavity. They lie closer together, too, on that side, and may even be anchylosed at their posterior extremities; while those on the other side, especially about their tubercles, describe an arch which, when the vertebræ are very much rotated, encircles the bodies of those bones. Moreover, they lie further apart; in fact, their posterior extremities are separated from each other in exact proportion to the amount of the axial torsion of the vertebræ. Hence arises this difference between the two halves of the thorax: that on the concave side of the spinal curve is narrower from before backward, but has greater capacity laterally, while on the side of the convexity it has the converse dimensions; and again, the perpendicular measurement is shortened on the former side, and lengthened on the latter.

The condition of the shafts of the several ribs accords with these facts, more particularly with that which has been last mentioned. On the side of the concavity they are rounded, or rounded and angular; on the side of the convexity they are unusually flattened and ribbon-like; but however likely it might seem from the appearance of the ribs, yet no difference can be detected by the most careful measurement in the length of the several ribs and cartilages of the two sides.

In angular curvature the chest has a different character,—that is, as I have already remarked, in angular curvature in the lower dorsal and upper lumbar regions. In the first place it is thrown upwards; the anterior extremities of the upper ribs rise considerably higher than the posterior, so that the upper, and still more the lower, ribs form an arch which is convex upwards. The consequence of this elevation of the chest is, that its perpendicular diameter is curtailed, whilst from before backward its dimensions are increased. The sternum preserves its relations to the bent column in the mesial line, but is thrown forwards; and as a consequence of the elevation of the anterior extremities of the ribs and of their arch-like vaulted form, it is thrown more forward the more it is depressed. The sides of the chest vary in shape according to the situation of the projection, and the acuteness of its angle. When it is in the lumbar region and moderately acute (94°), the ribs take a considerable lateral curve, and the chest is barrel-shaped; but if a projection in the same situation should form a more acute angle (60° for instance), and the column above, which includes the dorsal region, should be thrown much backwards; or if the projection should occupy the lower part of the dorsal region itself, the wrong direction of that part of the column in which the dorsal region is included, whether it be one leg of the angle or both, will give a different form to the sides of the chest: for those ribs which pass from the angular projection of the column will run a straighter course than the others, in order to reach the projecting sternum, and the sides of the thorax at that part will be flattened; while above the angle the chest will be vaulted in the way first mentioned. Should the angle be extremely acute (55°), the sternum will bend backwards at its lower end, and become slightly arched, as if its greater depression removed it too far for the ribs to reach it, even by their new and more direct course.

The thorax, in every instance, approaches very close to the pelvis, and may even rest upon it: the abdomen, therefore, is much shortened.

This, however, is a rule which the thorax does not follow in a very acute angular projection in the upper dorsal region, or when the curvature is situated low in the loins. For in a specimen of the former description, in which the nine upper dorsal vertebræ were destroyed, I found the ribs closely packed together at their posterior extremities, but considerably depressed and straightened anteriorly; so that the thorax was flattened laterally, its axis much inclined, and the sternum thrown forwards. The compensatory curvature of the column forward was so great that, in spite of the depression of the thorax, the distance between it and the pelvis was nearly natural. In an example of the latter kind, the thorax was elevated; but the very great amount of curvature forward in the remainder of the lumbar, and the whole of the dorsal regions, and the pendulous protrusion of the abdomen (compare the state of the thorax in curvature forward) contracted the perpendicular diameter of the chest, while the considerable vaulting of its walls laterally, enlarged its transverse dimensions. (Compare the remarks upon this case below in reference to the pelvis.)

In those cases of angular curvature in which the vertebræ are destroyed to a greater extent on one side than on the other, and in which, besides that the bones approximate on that side, it also happens, that the column above the angle is rotated on the axis of the vertebræ towards that side, and that the hump projects in the opposite direction; the thorax inclines towards the side on which the vertebræ are most destroyed, and sinks deeper on that side towards, and even into, the cavity of the false pelvis; while the sternum is depressed towards the same side as the hump; a compound, in fact, of lateral and angular curvature—scoliosis and kyphosis—produced by carious loss of substance, which may be named kyphosis scoliotica.

The state of the thorax is different again in curvature of the lumbar region forward, and in the excessive inclination of the pelvis backward, with which the lumbar curve coexists, and to which it is due. The chest is increased in length, and while the breadth of its lower part is strikingly greater than natural, it is very pointed above, and flattened from before backward. This remarkable form, especially the increase in length, is probably due to the abdominal muscles being stretched by the excessive inclination of the pelvis.

2. *Pelvis.*—The deformities of the pelvis in curvature of the spine are, in many respects, still more remarkable, and an acquaintance with them is, at the same time, more important. They are frequently the

primary deformity, and the spinal curvature is the consecutive; but even independently of these cases, they are not, as Meckel asserts, uncommon: on the contrary, they are so frequent, that it may be regarded as an exception to find a completely normal pelvis where there is any curvature of the spine.

In the great majority of cases the following deviations may be recognized with tolerable distinctness, and they may all be explained according to fixed rules.

The pelvis is always oblique in lateral curvature of the spine, and exhibits a want of symmetry in its two halves that is sometimes striking. The half of the pelvis on the side opposite to the upper or dorsal curve is higher than the other; the extremity connected with it appears shortened; that is to say, the several parts of the two limbs being of equal length, and the necks of the two femurs placed on a level with each other, the trochanter, the knee, and the heel on that side are higher than those on the other side: the elevated half of the pelvis inclines less than it should, and is, at the same time, narrow; the transverse diameter of the inlet, therefore, is greater than natural. The circumstances from which these changes appear to arise are twofold. In one respect they are due to a change in the position of the sacrum, which is both curved and rotated on its axis towards the side opposite to the lumbar curve. The consequence of the former—the curvature—is, that the os innominatum is somewhat lifted at the sacro-iliac joint; and the latter—the rotation—carries the posterior part of the bone into the cavity of the pelvis: the adjoining portion of the ilium follows; but, as the innominatum is fixed at the symphysis pubis, the linea arcuata bends near the sacro-iliac joint, and thus diminishes the capacity of this half of the pelvis in its antero-posterior diameter, at the same time elongating its transverse dimensions. But further, it is sometimes perceptible at the first glance, that the narrowness of this half of the pelvis is partly due to the flattening of so much of it as is anterior to the acetabulum. The reason of this flattening is found in the lumbar curve transmitting the weight of the body principally to the limb of that side. The linea arcuata then stretches in a straighter course from the angle already formed in it to the pubes, and the distance between the ilio-pectineal eminence and the promontory is diminished.

When, in consequence of combined lateral and angular curvature (Scoliosis kyphotica) in the dorsal region, the compensatory lateral curvature of the loins bends forward to a corresponding degree, the dimensions of the pelvis are narrowed still more; for the promontory, on the one hand, projects further into the upper aperture on the side of the curve, and as, on the other, the weight of the body is still more directly transferred to the neck of the femur on that side, the ilio-pectineal eminence is pressed inward and upward to a greater extent.

Observation of the character of the supplemental curvatures, therefore, enables us to predicate the deformity of the pelvis which exists in lateral curvature of the spine: the dimensions of its cavity are altered on the side opposite to the upper or dorsal projection, or on the same side as that to which the lower, or lumbar curve, or the spinous processes of the upper curve are directed.

Now and then, however, there are exceptions to this rule. Some very decided lateral curvatures of the dorsal region, which project considerably beyond the centre of gravity of the body, are continuous with a lumbar column which is, proportionally, much less curved; and the weight of the body, therefore, is transferred to that side of the pelvis which the dorsal hump overhangs. The innominatum on that side is then the higher, although the other changes which result in narrowing of the pelvis are still found, as in ordinary cases, on the side opposite to the dorsal curve. When beneath a lateral curvature to the right, in the upper part of the back, there is a curve in the lower dorsal and upper lumbar region to the left; and this is succeeded by a third deviation of the column in the lower part of the loins to the right, while the sacral vertebræ swerve again to the left; and when in such a case the second curve is greater than the rest, the left half of the pelvis will bear more of the weight of the trunk, and will stand higher, and have less inclination, than the right; while the deviation of the sacrum will give rise to the usual narrowing of the pelvis on the right side.

The pelvis, in angular curvature, is generally very capacious; its height is considerable, and the predominant diameter is manifestly the conjugate; the inclination varies, but usually—that is, when the projection occupies the usual situation in the lower dorsal, and adjoining lumbar regions—it is very decided.

This conformation of the pelvis chiefly arises from the diminution in the size of the abdominal cavity, which is produced by the depression of the thorax: the difference in its inclination depends especially upon the extent to which the compensatory curve projects forward. For the angular projection consists of two legs which diverge, under varying angles, from each other, one upward and the other downward. The greater part of the compensatory incurvation falls to the one or to the other of these legs, according to the situation of the projection; and the inclination of the pelvis varies with the distribution of the duty of compensation.

If the angle be situated in the lumbar region, the small remainder of the column in the loins, which forms its lower leg, is insufficient to counterbalance the projection backward, and there is no need of any further deviation in the pelvis, for the upper leg, which runs up to the dorsal part of the column, undertakes the compensation, and curves gently forwards. The inclination of the pelvis is then nearly natural.

The lower leg of an angular projection, situated in the inferior dorsal region, is longer and needs a more decided receding of the sacrum for its support. The inclination of the pelvis is then greater than natural.

When the angular projection is situated high in the dorsal region it is counterbalanced principally by curvature of its lower leg; and the sweep forward in the dorsal and lumbar region necessitates a still more marked inclination of the pelvis.

But, when the projection is in the lowest part of the lumbar region, the state of the pelvis is just the opposite. The rest of the vertebral column then forms the upper leg of the angle, the sacrum alone forms the lower. The pelvis is raised to a degree corresponding with the size of the angle, and the level of its upper aperture may even become horizontal; its inclination is annihilated, and the projection can be counterbalanced only by anterior curvature of the spinal column. If the number of vertebral bodies destroyed be considerable, it results as well from the loss of substance as from the elevation of the pelvis, that the trunk is shortened, and its two cavities narrowed; and a further consequence is, that the thorax acquires a peculiar shape. In the trunk of a woman, of 34 years of age, who died in childbirth, from rupture of the uterus, and who had had a difficult labor five years before, the spine was found projecting at an obtuse angle, in consequence of carious destruction of the bodies of the four lower lumbar vertebræ. The dorsal portion of the column from that point upwards, described a slight curve arching downward, while the sacrum, very flat and straightened, and constituting the lower leg of the angle, had raised the pelvis so much, that it had lost its inclination almost entirely, and that the distance of the upper margin of the symphysis pubis from the ensiform cartilage, was scarcely 3¾ inches. The antero-posterior diameter of the thorax was much contracted (compare page 187), so that the ensiform cartilage was not more than 3½ inches distant from the lower margin of the eighth dorsal vertebra, which was over against it: the transverse diameter, however, measured more than 9½ inches. The reason of this was that as the upper leg of the angle could alone undertake the compensation, the abdomen, being exceedingly narrowed from above by the anterior curvature of the column, and from below by the elevation of the pelvis, was thrust forward, and became pendulous, and the thorax was flattened from before backward, as in an ordinary anterior curvature in the lumbar region, by the same action of the abdominal muscles as resisted the expansion and sinking of the abdomen. It is worthy of notice that, without a close examination, so pendulous an abdomen might lead to the inference that the inclination of the pelvis was increased.

The ordinary anterior curvature in the loins involves a corresponding displacement of the pelvis backward, that is to say, excessive inclination of it; and more especially when the lumbar curve is itself compensatory and consequent upon too great inclination of the pelvis. When it arises from rickets it is always associated with diminution of the conjugate diameter of the pelvis: and when it is combined with lateral curvature also, the two halves of the pelvis are unsymmetrical.

But in some cases in which the lumbar vertebræ sweep forward into the pelvis, not only is there no increase in the inclination of the pelvis, but on the contrary, there is scarcely any, or none at all. Such cases are proved by what has been remarked already, and by the cause from which they generally arise, to be instances of angular projection in the lowest part of the lumbar region, which are compensated by anterior curvature of the upper leg of the angle.

§ 3. *Solutions of Continuity,—Dislocation,—Anchylosis.*—Various kinds of solution of continuity are met with in the vertebral column as results of external violence; and their characters are those of incised, punctured, or gunshot wounds, according to the instrument by which the injury was inflicted. The accidents to which the spine is most subject, however, are fracture of the bodies of the vertebræ, and laceration of the intervertebral cartilages. Though sometimes broken longitudinally, the bodies are much more liable to transverse fracture; very commonly one or several vertebræ are found comminuted, and the line of fracture runs in various directions. The injury which the spinal marrow sustains in these accidents usually renders them speedily fatal; but sometimes it may be observed, after death, that the fragments have begun to unite together by means of a scanty production of callus: it is extremely rare to meet with a specimen in which union has been completed. Fractures of the odontoid process of the axis present considerable interest; for in a few rare cases they have not only not proved fatal, but have even existed a considerable time without union of the fragments. A specimen of this kind is contained in the Vienna Museum.

The intervertebral substances are usually lacerated only when one or more vertebræ are at the same time broken or crushed.

The lateral articulations of the vertebræ are more rarely dislocated in proportion to their distance from the occiput and two upper cervical vertebræ.

Anchylosis is sometimes found in the spinal column at the time of birth, but it more frequently comes on later in life. The union takes place sometimes between the bodies of the vertebræ, the adjoining margins and surfaces of which are then connected together, and sometimes between their lateral articulations: it is also very common to find anchylosis in both situations. When the bodies are anchylosed, it may be by the union of their surfaces, which meet each other when the intervertebral substances have been removed by absorption or by inflammation and suppuration; or, it may be by a deposit of new bone at their margins, which passes, like a bridge, across the interspace between the bodies, and encloses the intervertebral substance in an osseous capsule; or, again, by a mass of bone (osteophyte), which seems as if it had been poured, when fluid, along the front of the vertebral bodies, and, then coagulating, had united them into one piece. Each kind of deposit forms transverse swellings between every two adjoining vertebræ: sometimes they grow, too, on the back of the column, and then they may prove dangerous, from their pressure on the cord (Key). Anchylosis of the lateral joints of the column may, of course, come on when the bodies are fixed in the manner just described; and it may take place between the atlas and axis, on the shortened side, in long-standing cases of wryneck. It may also result from inflammation and suppuration of the articular structures, from caries, and so forth.

§ 4. *Hyperostosis—Atrophy.*—Hypertrophy of the osseous structure of the spinal column never occurs to an extent at all to be compared with what is observed in the skull: sometimes only, in opening a vertebral canal, we may meet with some difficulty, in consequence, apparently, of the bony tissue being more dense than usual. Exostosis, too, is rarely found: when it does occur, its texture is generally cellular. If it spring from the back of the bodies of the vertebræ, and encroach upon the spinal canal, it may lead to serious consequences. The bridge-like osteophyte situated at the margins of the vertebral bodies, and that more abundant one which seems poured out upon them, are both commonly confounded with exostosis.

Atrophy of the vertebræ takes place, for the most part, only when there is general wasting of the whole skeleton. When it is a local affection, it is produced by aneurisms of the thoracic and abdominal aorta, and goes on to an extreme degree: the front and sides of the bodies of the vertebræ have, in some few cases, been completely eroded, and the spinal canal opened.

§ 5. *Diseases of Texture.*—Congestion of the bodies of the vertebræ is sometimes observed in the lower dorsal and lumbar part of the column, and it usually occurs when the vertebral plexus of veins is dilated and swollen; it is sometimes only an habitual distension unconnected with other disease; but more frequently it is produced mechanically by disease of the heart and lungs.

Inflammation of the vertebræ is a disease of frequent occurrence, not only in the young, in whom it is most observed, but also in adults. Indeed, old age is not exempt from it. Any portion of the column, or even any single vertebra, may be the seat of inflammation; but the parts mostly affected are the upper cervical vertebræ, and, next in frequency, the lower dorsal and the adjoining upper lumbar vertebræ. It is very commonly the primary disease, but sometimes it is brought on by previous inflammation and suppuration of the ligamentous apparatus of the column, and of the intervertebral substances. In most cases, it runs a chronic course, very often it is of tubercular nature, and terminates in caries and necrosis. When this is the case, matter usually forms and collects near the column, especially on its anterior surface; and, in favorable cases, opens externally: the track of the matter is sometimes very long, and the external opening far distant from the disease. The carious destruction may then be repaired by anchylosis, and by the column falling together at an angle, corresponding to the quantity of substance lost; but far more frequently the disease exhausts the patient, the symptoms usually showing that the spinal cord and its membranes suffer in some way or other. Thus the cord itself may be compressed from the tumefaction of the ligamentous apparatus, from the protrusion of an abscess into the canal, from dislocation of fragments, or of the whole, of a vertebra, or by the products of circumscribed inflammation of the dura mater of the cord; or it may be bent and irritated at the spot where the angular projection is beginning; it may waste, or circumscribed inflammation may take place in it, or diffused inflammation in its membranes, &c. Moreover, when the upper cervical vertebræ are carious, the odontoid process, being set free from its own ligaments, may, by a sudden turn of the head, tear through the inflamed and softened ligament and the dura mater, which confine it behind, and projecting naked into the canal, may crush the spinal marrow. When the upper dorsal vertebræ are carious, the abscess sometimes opens into one of the bronchi, and matter and necrosed fragments of vertebræ are discharged through the air-passage. Caries of the abdominal part of the column is very often combined with what is called psoas abscess.

Rickets in the spine may be distinguished by the peculiar change which it effects in the texture of the bones and by the curvatures produced in it by rickety deformity of the pelvis. As there is generally more distortion on one side of the pelvis than on the other, the curvature in the loins is usually directed forwards, and to one side.

It has already been remarked, that all the bones of the trunk are subject to *mollities ossium*, but especially those of the spine. It occasions different deformities, both of the pelvis and of the vertebral column, according to the particular condition of the patients. They are, for the most part, bedridden, so that the usual effects of the disease are an arched incurvation of the whole spinal column and elevation of the pelvis, produced by the pressure upon the sacrum and tubera ischii.

On the subject of *adventitious growths*, reference may be made to what has been already stated; those chiefly met with are tubercle and cancer. The former is of very frequent occurrence; it gives rise to extensive caries and necrosis of the column, and to the various consecutive appearances and terminations which have been pointed out as those of inflammation of the vertebræ. Cancerous deposits are more rarely met with in the vertebræ than in other bones. The same relations obtain, between cancer of the vertebræ and fungus, as it is called, of the spinal dura mater, as between the same disease in the cranium, and in the dura mater within the head: the former, when situated in the bones, may spread to the spinal dura mater, and the fungus of that membrane may reach from its original seat to the vertebræ, and become a fungus of bone.

THE THORAX.

§ 1. *Deficiency and Excess of Development.*—The full growth of the thorax is arrested in various degrees in monsters which are very incompletely formed; especially in those which are also acephalous or anencephalous, or which are born with spina bifida. The malformation of the thorax is in these cases associated with a corresponding deficiency, or a small and ill-developed state of the lungs and heart, and with hernial protrusion of those organs. It may arise either from partial absence of the spine, from fusion of some of the dorsal vertebræ together, from deficiency or incomplete development of the ribs, or from fission of the thorax and abdomen; and it consists in diminution of size and capacity, and generally, also, in misshapen exterior.

But, sometimes, persons, otherwise well formed, lack one dorsal vertebra, and one—generally the twelfth—rib; in a few instances, indeed, one or more ribs are found wanting, even when the number of vertebræ is complete. Occasionally, though there may be a proper complement of ribs, one of them is too short, and instead of reaching the sternum, it ends in a pointed cartilage.

Now and then there is no sternum, or only a part of it exists; or it is fissured at one spot, or in its whole length. The last condition is just indicated in some well-formed persons, whose ensiform cartilage is split or perforated, or in whom the lateral ossific centres in the body of the sternum remain separate for a long time. When the bone is wanting altogether, its place may be supplied, and the thorax closed by a firm fibrous membrane; otherwise, whether the opening be formed by a total or partial absence, or by a mere fissure of the sternum, the thoracic organs will protrude. Moreover, the bone may be unnaturally short, small, narrow, or the like.

In those double monsters in which the point of junction is the thorax, that part is found developed to excess. It is perternaturally developed likewise, when there is more than the proper number of ribs. Sometimes the supernumerary rib is borne upon a thirteenth dorsal vertebra, sometimes upon the first lumbar, while, in rare cases, the seventh cervical vertebra has a rib connected with it, which may either terminate by a free extremity, or become attached to the sternum, or to the true first rib. Some ribs are so broad as to appear double; others are fissured at different parts; while others, again, are forked at their anterior extremities, &c.

§ 2. *Deviations from the natural Size and Form of the Thorax.*—The chest is subject to several other varieties in *size*, or capacity, besides those which have been already mentioned: some of them are faults of original conformation, and are either connected with peculiarity in the general organization of the individual, or have a more immediate relation to some anomaly in the viscera of the trunk; while others arise from disease of the lungs, or pleura, or of the respiratory muscles. Whether the deformity be one of enlargement, or contraction, it may affect the two halves of the chest nearly or quite symmetrically, or it may be confined to one half, or even to a still less portion of it; but the change of form is greatest, when it occupies only a half, or a still smaller section, of the chest. The following are the principal deformities.

In some men the whole skeleton approaches, in its general form, the type which is characteristic of the female; and the thorax, as well as the pulmonary organs, are of small size. This individual peculiarity is the more marked, in proportion as the abdomen, more particularly, resembles the largely-developed abdomen of the woman. On the other hand, men are met with now and then, whose chests are disproportionately large and capacious.

Contractions of the chest, both when they are symmetrical, and when confined to one part, are observed in cases of pneumonia in which the lung is wasted, in catarrh and dilatation of the bronchi, in tuberculosis and tubercular phthisis in the lung, in pleurisy, and in paralysis and atrophy of the inspiratory muscles at the upper, anterior, and lateral parts of the chest, &c.

Dilatations ensue from emphysema of the lung, from pleuritic effusion, pneumothorax, and considerable effusions into the pericardium, from enlargement of the heart, dilatations of the aorta, large growths in the chest, &c.

The varieties in the *form* of the thorax present more interest than those of mere size, though the two are combined in numerous ways.

It is unnecessary to repeat, though it is desirable to refer to, what has been said, with regard to the deformities of the thorax connected with curvatures of the spine: the chief of those which remain are as follow:

The compressed and shallow chest, flattened from before backwards, which the clavicles and projecting shoulders overhang like wings, and which expresses the phthisical constitution. There is no question, that a thorax of this form is often associated with a peculiarity of the whole organization; but it is the latter, and not the form of the thorax, which predisposes to tubercular disease of the lungs. What such a chest loses in breadth and vaulted form, it gains in length; its capacity is by no means necessarily deficient, nor is that of the lungs within it; and that such a form of chest gives a predisposition to phthisis, is quite hypothetical.

The thorax is sometimes flattened, or even depressed, in the subclavicular regions, and thus obtains the phthisical form. This change is a consequence of wasting of the pulmonary tissue in the vicinity of tubercles, or of closure of vomicæ in the apices of the lungs: it also sometimes results from pleurisy in the same region.

In general emphysema of the lungs, the chest is enlarged, and acquires a vaulted barrelled shape.

After the subsidence of chronic inflammation of one whole pleura, the corresponding half of the chest becomes flattened; or it may even sink in and form a kind of pit. If the pleurisy have been confined to a part of the membrane, a similar deformity takes place, but occupies only that portion of the chest. In the former case the ribs fall in, especially at their anterior extremities, and lie so close together along their whole length, that the interspaces between them are obliterated; and the diameter of the chest is diminished on that side in every direction. The consequence is, that the dorsal vertebræ curve towards the opposite or sound side of the chest, and that a curvature of compensation takes place in the lumbar region; at a later period of life it may even be found that the deviation of the spinal column has reached the pelvis and rendered it oblique.

There is a remarkable deformity of the chest which is known by the name of pigeon-breast. The cause of it is an attenuated condition of those muscles of inspiration which are situated at the upper, anterior, and lateral regions of the chest, viz., the pectorales and serrati; and it is very frequently, though not constantly, combined with rickets of the thorax. The chest is flattened laterally; and very frequently it has even a longitudinal depression towards the anterior extremities of the bony ribs, while the sternum, with the costal cartilages, strongly curved, projects considerably in front (pectus carinatum). The spine is either straightened, or presents a slight excurvation. The diaphragm becoming hypertrophied, carries on the respiration, and makes up for the diminished breadth of the chest by increasing its vertical diameter. The depression of the diaphragm considerably augments the size of the abdomen externally: and as the latter circumstance has chiefly arrested the attention of observers, it has led to numerous misconceptions as to the true theory of the disease. The ribs frequently bear the clearest marks of rickety disease of their tissue; and even in life the enlargement of their anterior extremities may be easily perceived.

Another, and an important deformity of the thorax, is that in which it becomes narrowed and cylindrical, and, at the same time, elongated. The change arises from paralysis of the intercostal muscles, and is produced by enlargement of the inferior intercostal spaces. It has been named by Engel the paralytic thorax (Oesterr. Jahrb., April, 1841).

The contraction of the thorax which takes place in old age is sometimes allied to one, and sometimes to the other of the two last-described deformities.

The chest sometimes becomes misshapen in consequence of following certain employments and trades: thus shoemakers have a depression at the lower end of the sternum.

Amongst the peculiarities of the several parts of the chest, there are still those of the ensiform cartilage which require notice. The most remarkable is the inversion of a long processus ensiformis.

§ 3. *Solutions of Continuity.*—Fractures of the ribs are of serious moment, from their sometimes injuring the pleura and lungs, and even the pericardium and the heart. Single fractures, for the most part, unite readily, but when several successive ribs are broken, false joints are sometimes formed between the tumors of callus thrown out around their fragments. Fractures of the sternum are usually transverse; they seldom occur unless the ribs or spinal column are injured at the same time.

§ 4. *Hyperostosis, Atrophy.*—The spongy exostosis sometimes presents itself on the ribs and sternum as an example of hyperostosis. Atrophy of the ribs is commonly well marked in tabes senilis of the skeleton; and both ribs and sternum are frequently worn away, and even perforated by the pressure of aneurisms of the ascending part, and arch of the aorta.

§ 5. *Abnormal changes of Texture.*—Caries and necrosis are frequently met with in the ribs and sternum. They are often produced by empyema and suppuration in the pleura, by the softening of tubercular lymphatic glands, by vomicæ of the lungs arising from tubercle, &c. The inflammation and induration which occur in syphilitic disease are rarely met with in the sternum.

Morbid growths are rather frequent both in the sternum and ribs; and they present the various forms of enchondroma, tubercle and tuberculous caries, and of cancer; the last may be a consequence of the degeneration of that disease in the adjoining glands of the chest, or a substantive new growth in the bones themselves.

THE PELVIS.

§ 1. *Deficiency and Excess of Development.*—There are various ways in which arrest of development is manifested in the pelvis. Sometimes the sacrum and coccyx are defective, or altogether absent, or they are stunted in their growth. At other times, one or both of the innominata, or a part of one of them, is wanting, there being at the same time no lower extremities; or a fissure existing in the abdomen may be continued down to the pelvis, and lay open the symphysis. In the siren-monster the lateral parts of the pelvis are fused together.

Allied to this is a pelvis which is of diminutive size, either from its wn original conformation, or in consequence of defective growth of the xual organs or rectum.

Excess of development is exemplified in double monsters, in whom it eaches various degrees.

§ 2. *Deviations of the Pelvis from its natural Size and Form.*— mongst the former are included specimens in which the pelvis is unaturally large or wide in all its diameters, as well as those in which it unnaturally small or narrow in the same respect. As pelves, which re unusually large or small in any one diameter, are generally in an pposite condition in some other dimensions, they will be treated of under he head of deformities. And small pelves, more especially, not only ear traces of their growth having been arrested in consequence of ickets, but they are, at the same time, also misshapen. We shall meet ith various instances of such pelves amongst the following deformities. he essential characters of a rickety pelvis are, that it is small, i. e. low nd contracted, especially in the conjugate and oblique diameters of the let; its capacity is small, its inclination considerable, and the arch of he pubes is widened. When this fundamental anomaly is extremely eveloped, or unequally on the two sides, it will be specially noticed as f rickety origin when it occurs in the following description.

As the importance of the subject of deformity of the pelvis arises hiefly from its connection with the mechanism of parturition, the prinipal interest is centred in the upper aperture or inlet. The great umber of facts relating to the subject can best be arranged according o Osiander's division of deformed pelves; but, as that division does not nclude every deformity, it must be somewhat enlarged, by the addition f several subinordinate varieties, and by the interpolation here and here of an intermediate form.

Osiander enumerates six forms.

1. That in which the pelvis is elliptical in its transverse diameter; he ilia are widely separated from one another, and, as on the one hand, he promontory of the sacrum encroaches a little forward, and, on the ther, the pubic bones are flattened, the conjugate diameter is less than atural, and the transverse measurement increased.

2. The kidney-shaped pelvis, in which the great projection of the pper part of the sacrum produces a deformity of that figure at the nlet.

There is a form of pelvis intermediate between these two, in which he base of the sacrum runs straight across the back of the inlet, and the inea arcuata bends forward from it at an angle.

3. That pelvis, the upper aperture of which may be compared to a igure of ∞. The extreme projection of the promontory on the one ide, and the sinking backward of the symphysis and horizontal rami of he pubes on the other, produce a deformity in which the upper aperture ppears divided into two lateral spaces, which are united by an interening isthmus.

These deformities of the pelvis, with scarcely an exception, are always ccasioned by rickets.

4. The pelvis, which is oval or elliptical in its conjugate diameter, the

antero-posterior measurement exceeding the transverse. I have met with a deformity of this kind combined with angular projection of the spine.

5. The oblique pelvis. This class includes by far the greatest number of misshapen pelves. Its characters are as follow: the ileo-pectineal eminence approximates unnaturally to the promontory on one side; that half of the pelvis has a diminished oblique diameter, it stands on a higher level, and inclines less than the other. The causes which lead to such a change of form are lateral curvature and torsion of the sacrum, and straightening and encroachment inward of so much of the linea arcuata as lies between the acetabulum and symphysis. It is a class which includes those frequent deviations from the natural form of the pelvis that arise from lateral curvature, and yet more frequent primary deformities induced by rickets, as well as those which result from dislocation of one hip, whether in consequence of violence or from previous disease of the joint. Of the last, which is the most common cause, I shall speak further presently.

The pelvis is sometimes oblique at the time of birth, but much more frequently it becomes so afterwards. There is much interest attaching to those congenital obliquities of the pelvis, which originate in faulty formation, as contradistinguished from those which are produced by disease in the fœtus. One of these, in particular, has been described by Nägele, under the name of the obliquely contracted, or obliquely oval, pelvis.

The deformity so named, is founded in congenital anchylosis of one sacro-iliac synchondrosis, and in arrested growth of the lateral mass of the sacrum, and of the ilium on the same side.

The principal other characters of the obliquely contracted pelvis arise out of these two; and in two specimens in the pathological collection at Vienna (in both which the anomaly is on the right side), they are seen to correspond in all particulars with Nägele's lucid description: they are as follow:

The sacrum appears displaced towards the anchylosed side, and its anterior surface is turned more or less in the same direction; the symphysis pubis is driven towards the other side, and therefore lies obliquely, not directly, opposed to the promontory.

The lateral wall of the pelvis on the side adjoining the anchylosed part is flatter and straighter than natural.

On the other side, the line which separates the greater from the true pelvis, is less curved than usual in its posterior half, but more so anteriorly; this anomaly, together with its remote consequences, is often found also in obliquities that come on after birth.

Hence it follows, that the pelvis is obliquely contracted from the sound sacro-iliac joint to the opposite acetabulum, whilst the measurement between the anchylosed synchondrosis and the acetabulum, on the healthy side, is not only not diminished, but is greater than usual:

That there is less distance on the side of the anchylosis than on the sound side, between the promontory and the vicinity of the acetabulum, and between the point of the coccyx and the ischial spine:

That the posterior superior spine of the ilium is further removed from he lower border of the symphysis pubis on the anchylosed than on the rticulated side:

That the walls of the pelvic cavity converge somewhat at their lowest art in an oblique direction, and that the arch of the pubes is more or ess narrowed:

And that the aspect of the acetabulum on the flattened side of the elvis inclines rather forwards, while on the other side it looks almost lirectly outwards.

In the specimens preserved in the Vienna Museum, the lumbar part f the vertebral column is distinctly curved towards the abnormal side f the pelvis.

There is another form of unsymmetrical pelvis, which is allied to the receding, but subordinate to it in degree, and generally indeed is only ust discernible. The deformity is found in persons in whom the last umbar has on one side been converted into a sacral vertebra. The inquality in the pelvis consists in its greater capacity on the side of the nomaly in lumbar vertebra, the linea innominata of that side describing larger and shallower curve, and being more inclined than its fellow. The opposite half of the pelvis is in the contrary state; and with that state is combined a slight curvature and rotation of the vertebræ towards he less capacious side,—an anomaly which is remarkable for the analogy t bears to the condition of the pelvis in lateral curvature, and which is onstantly found in the lumbar region of the spine whenever its last ertebra is thus converted into a sacral. In cases in which this peculiarity in the last lumbar vertebra exists on both sides, the projection of he promontory is very slight, the conjugate diameter is great, and the nclination of the pelvis considerable.

Almost all the obliquities of the pelvis which come on after birth are f rickety origin. We have seen that when the sacrum, and consequently he pelvis, incline too much backward, and the latter is at the same time arrowed symmetrically, a compensatory curvature forward, a genuine ordosis, takes place in the lumbar region. In the same manner, when ne side of the pelvis is contracted in consequence of a deviation of the acrum from its mesial direction, the anterior curvature in the loins nclines somewhat laterally towards the contracted side. Thus the bliquities of the pelvis, in cases of rickets, are at once the starting-oint and the occasion for lateral, or more commonly antero-lateral, urvatures in the loins; and these are succeeded by supplemental deviaions of the dorsal part of the column towards the opposite side and ackwards. Such is the state of the spine when the deviation is a compound one; and the conclusion to which an analysis of it leads is the ame, so far as regards the deformity of the pelvis, as that which was licited by an examination of the condition of the pelvis in primary lateral curvature: in both cases, the abnormal half of the pelvis is on the side opposite to the dorsal curve of the spine.

6. *The triangular pelvis.*—When the amount of this kind of deformity is slight, the inlet of the pelvis forms a triangle, with its angles rounded off, and the sacrum for its base. In a higher degree of it, the sides of the triangle become convex, and encroach upon the pelvic cavity, and after a time the base projects inward too; the three sides then unite at

very acute angles, and the upper aperture of the pelvis acquires the shape of a heart on playing-cards. The deformity may even go farther; both sides of the triangle, or it may be one only, approximating to the base so much as to bring the promontory into contact, and even into union with them, just above the acetabula.

The triangular pelvis is the extremest instance of contraction. The ilia are so compressed from before backward that their venter becomes a narrow fissure; the symphysis pubis forms a beaklike prominence; the arch of the pubes narrows considerably, and may even be obliterated; the tubera ischii approach each other; and as the promontory and the lumbar part of the spine sink down into the cavity of the pelvis, the sacrum beneath presents a curved anterior surface, or may be bent at an angle.

The triangular pelvis is, for the most part, a result of mollities ossium; but it is an error to ascribe it exclusively to that disease. Mollities ossium may undoubtedly be the cause of every decided and advanced degree of triangular pelvis, but minor degrees of it are sometimes due to rickets. In extreme cases there is less inclination of the pelvis than usual, and it is sometimes even raised above the horizontal line.

The form and position of the pelvis undergo an interesting change after so-called coxalgia: for the dislocation of the femur upward and backward which takes place in that disease, when it occurs only on one side, destroys the symmetry of the pelvis in a remarkable manner, and renders it oblique; and the same change occurs when the dislocation is the result of violence. The general characters of the deformity are, that the whole os innominatum becomes wasted and small, the ilium assumes nearly a vertical direction, the upper aperture becomes enlarged, and the pelvis acquires an abnormal inclination. In other respects its characters present many varieties which may depend upon the extent of the displacement, upon the condition of the joint as to mobility or anchylosis after the diseased process has ceased, or supposing the limb to have been movable, upon its having been used afterwards or not.

a. In cases of coxalgia on both sides, in which a layer of more or less dense fibrous tissue binds a remnant of the head of the femur, or the stump of its neck, to the upper margin of the wasted acetabulum, and in which, therefore, some mobility of the limb remains, the ossa innominata are attenuated, especially at the pubes and ischium, and the whole cavity of the pelvis is enlarged. The latter change is partly due to the attenuation of the bones; but it can be traced, at particular parts of the pelvis, to other circumstances. The inlet is enlarged by the curve of the linea innominata becoming shallower, and by the disappearance of the iliopectineal eminence, and of the angle at the sacro-iliac synchondrosis; the cavity of the pelvis increases in size symmetrically, at the expense of the basis of the wasted acetabula; and the very striking dilatation of the outlet is due to the separation of the ischia from each other. This separation of the ischia is attributed by Hülshof to the action of those rotating muscles of the femur which arise from the tuber ischii; for as the whole weight of the body falls on them, they draw the ischia outwards, and consequently, asunder. The angle beneath the pubes thus becomes obtuse and may even be changed into a shallow arch. The ilia assume nearly

al position, partly in consequence of the abdominal muscles at-
to them being stretched by the sinking of the pelvis, and perhaps
om the pressure of the displaced head, or stump of the femur,
the upper part of their outer surface.
wasting and enlargement thus described, diminish the height of
.vic cavity, and the length of its axis; the pelvis itself is de-
, and has a greater inclination than natural, and the lumbar part
pine forms an arch, directed forward, which varies in degree, ac-
; to the amount of the inclination of the pelvis.
ı cases in which similar changes have been produced by coxalgia
rticular structures of the hip, but on one side only, the os inno-
m of that side becomes thin, and in consequence of some flattening
king of the linea arcuata, and of a distortion of the sacral and
vertebræ, which has yet to be described, the inlet of the pelvis is
d on the same side. The capacity of the pelvis is also increased,
size of the outlet varies, according as the limb connected with
ased joint has been used or not. If it have been used, the cor-
ling tuber ischii projects outward, and the dimensions of that side
utlet are increased; but if not, the weight of the body, in walking
nding, will have been sustained, in whole or in part, by the sound
hile the actions of the diseased joint will have been assisted, or
pplied, by the use of a stick or crutch. The tuber ischii, in that
not everted; but on the contrary, the wasting which accom-
the process of repair, the shrinking of the acetabulum, the an-
ending of the os innominatum, and, in many cases, the inward
n which is given to the femur while the patient is lying in bed, and
s afterwards retained, all tend to force the tuber ischii inward,
lly towards the coccyx, and thus to contract the outlet of the
n the side of the disease.
over, the ilium on this side becomes so nearly vertical, that the
e between its anterior superior spine and the upper border of the
sis pubis, is manifestly lessened: the height of the same half of
is is diminished; it is depressed, that is to say, its inclination is
than that of the other side; and the sacrum is both inclined back-
n the side of the diseased joint, and rotated towards it. The two
of the pelvis, therefore, are not symmetrical; and the error of
try will increase in proportion as the acetabulum of the sound
driven inwards and upwards, to receive the weight which the
spinal column directs upon it,—in proportion, that is, as the
becomes contracted on the sound side, while it is enlarged on the
d. The difference between the two sides will increase, too, so long
bones continue soft.
he curvature of the vertebral column, which has been referred to,
he purpose of compensating for the obliquity of the pelvis, it is
d forward and towards the side opposite to the coxalgia, that is,
the healthy, or narrower, half of the pelvis. It is a curvature
compensates for the deviation of the sacrum, and if it be itself
rable, it is followed by a curvature in the dorsal region towards
osite side. In this, again, we find a correspondence with the rule
wn as to the relation of deformities of the pelvis to primary lateral

curvatures, that the pelvis is contracted, &c., on the side opposite to the dorsal curve.

γ. When the morbid process has ended in complete anchylosis, the linea arcuata and the bottom of the acetabulum, on the diseased side, become not only flattened but angular; in fact, the whole os innominatum, wasted in all its separate parts, bends at an angle projecting outward, which is formed in the seat of the old acetabulum,—in the osseous cicatrix corresponding to the previous disorganization. The ilium inclines inward and forward, the ischium inward and backward, the promontory encroaches on the healthy side of the pelvis; and as the symphysis pubis is drawn by the angular distortion of the innominatum towards the side of the disease, the linea arcuata of the healthy side runs in a straight direction forward to reach the displaced symphysis.

δ. Under certain circumstances, which have not as yet been clearly ascertained, the pelvis is found to have all the anomalies, already mentioned, as results of coxalgia, but to be elevated, and to have less inclination on the diseased side; although, at the same time, the extent of the consecutive luxation is not greater than that which has been presumed in the foregoing description, nor has the process terminated in a different manner. (Compare Guérin.)

ε. On the other hand, it certainly happens after complete dislocation upward and backward upon the outer surface of the ilium, whether it be the result of violence, or have occurred in the course of coxalgia, that the pelvis is raised, and has less inclination on the diseased side. This peculiarity was attributed by Guérin to the action of the stretched psoas and iliacus muscles: and there may be observed, at the base of the anterior inferior spine of the ilium, a more or less distinct impression or furrow, which has been made by the pressure of the common tendon of those muscles. The same condition is found when the disease terminates in anchylosis, after having destroyed the head and neck of the femur. The small trochanter is drawn upward against the border of the acetabulum, and is also turned backward by the inversion of the limb.

Besides these deformities of the cavity and the outlet of the pelvis, which either accompany, or may be inferred from the abnormal states we have already considered, there are several others of independent origin. They depend chiefly upon the sacrum, whether it be too much flattened or too much excavated, upon unusual projection of the coccyx inwards, upon excessive width or narrowness of the arch of the pubes, &c.

§ 3. *Deviations from the healthy Condition of the Articulations of the Pelvis, and Solutions of the Continuity of its Bones.*—There are various circumstances under which the synchondroses are liable to become more or less loose. Not only may they be torn asunder by considerable mechanical violence, but, in pregnancy, the firmness with which they connect the bones is slightly diminished, the fibro-cartilages becoming succulent, soft, (and vascular?): in the act of parturition the fibro-cartilages may be very much stretched, and even partially separated from the bone. When puerperal diseases of a very malignant type come on after labor, the cartilages may be partly or entirely removed by the destructive suppuration, and the bones thus separated from one another.

On the other hand, the bones of the pelvis may be too closely connected together, and anchylosis may take place between them. It is usually effected by bridge-like processes of bone which pass from the margin of the articular surface of one bone to that of the other, and as it were enclose the fibro-cartilage in a kind of capsule; it very rarely happens, that there is any union of the articulating surfaces themselves; and it is not ascertained whether, when that is the case, the fibro-cartiages themselves ossify, or whether, as is more probable, new osseous matter is formed on the articulating surfaces of the old bone, whilst the ibro-cartilage is absorbed. Anchylosis of the sacro-iliac joint is met with now and then, but it is rarely found at the pubes.

Fractures of the pelvis do not generally take place without very considerable external violence, such as a fall from a great height, being run over, or buried beneath falling earth. They seldom unite without permanent displacement of the fragments.

§ 4. *Hyperostosis, Atrophy, and Diseases of the Texture of the Bones of the Pelvis.*—With the exception of those osteophytes which form on the pelvis in consequence of inflammatory processes, or caries in the hip-joint, we find hyperostosis but rarely in the pelvic bones; whilst atrophy, especially of one half of the pelvis, is frequently met with after coxalgia and the several dislocations of the femur.

Caries and necrosis occur in the pelvis, chiefly as consequences of disease in the hip-joint; they arise sometimes also from the pressure of the bed, from the suppuration of lymphatic glands, muscles, &c.

Rickets and mollities ossium occasion the deformities of the pelvis already described.

On the subject of adventitious growths it may be mentioned, in addition to what has been stated already, that the caries which comes on in the bones of the pelvis is frequently of tubercular nature; and that cancer occurs in them not only in the form of primary tumors, which sometimes attain a great magnitude, and have an abundant bony skeleton, but also, secondarily, by the advance of cancer from the rectum, uterus, or vagina.

OF THE EXTREMITIES.

§ 1. *Defective and Excessive Development.*—The former class includes those cases in which one, or more, or all of the extremities, or some part of one of them, is wanting, or in which their development is arrested. In the upper extremity it may happen that there is no humerus, or that one or both of the bones of the fore-arm are absent, or, if present, are in a rudimentary state. The hand will then be found articulated to the humerus, or to the scapula, according to the special deficiency of the intermediate bones. The hand itself may not exist at all, or only a part of it may be developed. In the case of partial deficiency of the hand, a correspondence is observed, both in the carpus, the metacarpus, and the fingers, with the character of the defective development in the fore-arm: when the radius is wanting, the thumb and forefinger, with so much of the carpus as belongs to them, are wanting too; and the other fingers, and their carpal elements, do not exist when there is no ulna; but when either bone of the fore-arm is in a rudimentary state some trace of the corresponding part of the hand, an incomplete finger, for instance, can also be found. Again, the hand may terminate at the metacarpus, or in one or more incompletely formed fingers; and, lastly, in some cases, a fusion of the bones of the metacarpus and fingers is observed.

Deficiencies of the same kind occur in the skeleton of the lower extremities. Only in them a peculiar fusion of the bones takes place, which is known by the name of the Siren monstrosity. The bones, in this case, are also rotated on their axes forward.

The chief instances of excess of development are those in which supernumerary fingers and toes, or the last phalanx of an additional finger or toe exist; they occur in persons who may be otherwise well formed. The development of additional long bones and limbs, whether complete or incomplete, indicates a tendency to duplication in the whole body.

§ 2. *Morbid Varieties in point of Size.*—Under this head may be included that disproportion in length affecting all the limbs, or the thoracic or abdominal extremities only, which dates from the period of their original formation; it is of most importance when it affects the bones of a single extremity or of one segment of an extremity. Thus the humerus or one of the bones of the fore-arm may be found shortened in a remarkable degree, and in the latter case some anomaly will exist in the articulation of the carpus, &c., to the fingers and toes: sometimes a bone exceeds its natural growth, and reaches a monstrous and disfiguring size.

A bone is liable to an interruption of its growth at any period, or it may be the seat of atrophy; in either case, the whole, or part, of an extremity, will present an instance of acquired smallness of size. Instances of this kind are frequently met with after exhaustive processes of disease and repair, either in bones or in the soft parts adjoining them, after inflammation and suppuration in them, fracture, caries, necrosis, and rickets, after unreduced dislocations, neuralgia, paralysis, &c.

§ 3. *Deviations of Form.*—These are very numerous, independently of those which arise from defect or excess in the original formation of the bones. The bones are variously misshapen in dwarfs, in cases of hyperostosis, after the loss of the substance produced by caries, from osteoporosis, &c.; and their form is altered still more after fractures which have united with the fragments displaced, and after permanent dislocations; but the most remarkable deformities of all are the various bendings and curvatures of the long bones which are produced by rickets, &c., especially in the lower extremities.

§ 4. *Solutions of Continuity.*—Every form of solution of continuity is exceedingly common in the bones of the extremities, but fracture is the most so. The whole subject has been considered in general already, and it only remains to treat in particular of fractures of the neck of the femur and of the patella.

Fractures of the neck of the femur are, for practical purposes, divided into those within the capsular ligament and those external to it. The former may occupy any spot intermediate between the head of the bone and the insertion of the capsular ligament. Its plane is sometimes transverse, but more commonly it is oblique: in the latter case, it either runs through the base of the neck near the insertion of the capsule, or, which is more frequent, passes somewhat further out and traverses the great trochanter.

Moreover, the two kinds of injury may be complicated together, the same fracture being partly within and partly without the capsule; and again, there may be one fracture within the capsule, and a different one external to it, running through the trochanters.

The diagnosis of these factures has for a long time engaged much of the attention of surgeons; but the fracture within the capsule has been the chief subject of investigation, with reference to the question of its reuniting by means of bony callus.

So rarely is this fracture reunited by bone, that many have doubted the fact; and on this account, as well as from the frequency of the accident, it is important to be acquainted with the changes which, in different cases, take place in the fragments.

It is often observed in very decrepit persons, that even after a long period no trace of inflammation and exudation is discoverable upon the fractured surfaces. The capsule is slightly reddened and swollen; but the only change in the surfaces of the fragments is, that they are smoothed off in a marked degree by absorption. There are some cases in which, though the fragments present no mark of reaction, they are absorbed to such an extent, that the head of the femur forms a flattened or concavo-convex disk, and its neck has almost entirely disappeared.

Sometimes partial necrosis takes place in the fragments, and the subsequent reaction degenerates into suppurative inflammation of the bone and articular capsule, and caries within the joint.

Usually the fragments, thus diminished more or less in size by absorption are covered with a fibroid (ligamentous) tissue. This covering is, in fact, callus, arrested in the progress of its development to bone, but in which now and then a few isolated splinters, or needle-like growths of new bone, do form. The fibroid tissue occasionally serves to bind the fragments closely together; but more commonly it is drawn out into ligamentous cords, which are inserted into the margins of the fractured surfaces and compose altogether a tolerably complete capsule: such a capsule connects the fragments but loosely together, and their surfaces are movable over one another. Or, again, there may be no ligamentous bands formed, and the surfaces of the fragments, unconnected with one another, but covered with the fibroid exudation, may articulate together within the old capsule. The false joint resulting has a freedom of motion proportioned to the quantity of the fragments which has been removed by absorption.

The rubbing of the surfaces against one another gradually wears down their fibroid covering of callus; it gradually, also, renders them smooth, and produces an ivory-like condensation of their spongy tissue. Hence, when the covering is entirely removed, two bare, smooth surfaces of bone, glistening and polished like ivory, are left to articulate together.

The false joint thus produced varies in its form. Sometimes two tolerably flat and even surfaces are applied to one another; at other times, one of the fractured surfaces is slightly hollowed out into an articular cavity, and the other forms an articular head. Accordingly, at one time the stump of the neck is rounded off, and fits into the fractured surface next the head, which has been a little excavated; while at another time, after absorption of the neck, the head of the bone moves in a large and slightly concave articular surface, hollowed out between the two trochanters. It is worthy of remark, that in the latter case the line of the insertion of the capsule recedes to an extent corresponding with the absorption of the neck, and that the size of the articulation is thereby increased.

In a specimen of fracture of the neck of the femur, in the Museum at Vienna, a fragment of the head of the bone having fallen upon the fractured surface of the neck, and become bound to it by ligamentous tissue, articulates by its external and still cartilaginous surface, with the fractured surface of the rest of the head.

The fragments are in some cases, uneven and serrated, and become wedged into each other at the time of occurrence of the fracture; their surfaces then unite together by fibroid callus.

In a more frequent instance of this kind of accident, the neck of the bone becomes implanted in the spongy tissue of the great trochanter.

While these changes are going on in the fragments of the bone, the capsule of the joint becomes swollen, and its contents more or less turbid: it also frequently forms adhesions, of a cellular or ligamentous nature, with the fibrous investments of the neck of the bone, as well as with the fibroid callus upon the fractured surfaces.

In some extremely rare instances the fragments do unite within the capsule by means of bone; it is a mode of union which is almost always very slowly accomplished, and in which there is considerable shortening of the neck of the bone. That fracture, too, in which the neck of the bone is driven into the spongy tissue of the trochanter has been seen repaired by bony callus.

The rarity of union of intra-capsular fracture of the neck of the femur by bone, when the accident is so frequent, has for a long time elicited much research into the reasons why the customary mode of repair does not take place in this instance. Many reasons have been assigned for it, but it must be acknowledged that none of them is satisfactory. Amongst them are the following:

α. In general, the advanced age of the persons in whom the accident occurs; and, in particular, a state of atrophy of the skeleton.

β. Insufficient nutrition of the separated head of the bone, by the few vessels that enter it through the ligamentum teres.

γ. The presence of synovia and other effusions between the fractured surfaces.

δ. The difficulty of securing and maintaining proper adaptation of the fragments to each other, and the want of some permanent pressure to effect it: and, lastly,

ε. Want of rest.

No one of these reasons, as have been stated, sufficiently explains the matter. That no reaction takes place in the fractured neck of the femur n an aged and decrepit person is intelligible, because the same fact is very often observed in such persons in fractures elsewhere: but this does not explain why the formation of callus should be so backward generally, even under less unfavorable circumstances, and why the proper changes in it should be so commonly frustrated. This consideration derives additional importance from the fact, that in the majority of cases, and even as a rule, no bony union takes place in fractures of any bony structures which are enclosed within an articular capsule.

On more closely examining the subject, the principal conditions appear to be two: the first is, that no primary or provisional callus whatever is formed: and the second is, that the secondary or definitive callus is arrested in its development at the stage of fibroid (ligamentous) tissue. It is the want of the first callus that, for the most part at least, arrests the growth of the later callus, and commonly leads to the formation of a false joint. The only object now, therefore, is to determine the reasons why no provisional callus is thrown out.

The following circumstances appear to me to be the chief impediments:

1. The small amount of vascularity possessed by the portion of the fibrous capsule of the joint, which occupies the place of periosteum; to which, as well as to the density of its structure, and, more especially, to its close adhesion to the bone, it must be attributed, that no exudation, preliminary to the formation of the provisional callus, takes place between the bone and its fibrous investment.

2. But the principal cause is the small share which the soft parts take in the reactionary process: they are placed at some distance from the seat of fracture; and the distance is further increased by distension of the capsule with exudation: in addition to this it happens that the exudation, whether in consequence of its being diluted with synovial fluid, or from its original quality, almost never becomes organized to bone.

It is, therefore, the want of the first callus and of that firm fixing of the fragments which should be effected by it, that really prevents any union of the fragments by bone, that interferes with the production of the secondary callus, and leads to the formation of a false joint; and the influence of this deficiency is, in ordinary cases, greater in proportion as the difficulties enumerated under the head δ and ε are brought into action.

However constant the failure in the production of callus at the seat of fracture itself, yet now and then the growth in question does occur in other places, when an energetic reactionary process springs up around. Thus, sometimes shallow cup-like masses of callus are developed on the enlarged capsule of the joint; in other cases, especially those in which the neck of the femur is driven into the substance of the trochanter, callus is poured out upon and around that process; and in some other rare cases the head of the bone unites with the acetabulum by a deposit of callus, which produces atrophy of the cartilage covering the bones, and takes its place.

Fracture external to the capsule is generally repaired by bony union, but sometimes a false joint is formed. Even in the former case, the union is attended with some deformity, either displacements of the fragments and shortening of the extremity, or shortening, and a more horizontal position than natural of the neck of the femur. Sometimes, too, the neck is driven into the spongy tissue of the upper end of the femur.

Fractures of the neck of the femur, as I have already mentioned, are of frequent occurrence. The principal predisposing causes are advanced age and senile atrophy of the bones. To these may be added, the depression and more nearly horizontal direction, which is peculiar to the neck of the femur in old age; and as that position of the neck is the normal condition in the female, it is supposed to account for the greater frequency of the accident in the female sex. The external cause is usually a fall upon the trochanter, or a fall vertically upon the feet and knees.

Transverse fracture of the patella requires notice, because of the considerable displacement which attends the repair of the injury. The capsule of the knee joint is enlarged upward on the femur to a corresponding degree.

§ 5. *Disease of Texture.*—Like hyperostosis, these diseases have been sufficiently described in the Chapter on Diseases of the Bones in general.

PART X.

ANOMALIES AND DISEASES OF CARTILAGES.

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Cartilages are naturally divided into the true and the fibrous, and ccordingly the morbid affections of each kind require to be separately oticed.

§ 1. *Deficiency and Excess of Development.*—An absolute primordial eficiency of these structures is seen only in concurrence with the entire bsence or partial development of organs, into the composition of which ey enter.

So, too, an original excess of their development is very rarely met with, nless those parts, of which they form the ground-work, be more or less mpletely double. Now and then, however, some small supernumerary ieces of cartilage are observed on the external ear, the nose, and the rynx, and especially at the inlet of the last-mentioned part.

But during extra-uterine life, the production of cartilage is a less nusual occurrence. It approximates in the character of its texture metimes to true cartilage, and sometimes to fibro-cartilage. Many of e false joints formed in cases of ununited fracture present instances of is growth in the white substance which invests the broken ends of the one: cartilage is developed in many of the tendons, and in that situaon it usually ossifies; the enchondroma, which is met with in the bones d different soft parts, affords another example of the same kind, &c. is, however, necessary to remark, that many productions which have en hitherto reputed cartilaginous, or fibro-cartilaginous, are discovered y closer examination not to belong to either class: such, for instance, the majority of the so-called loose cartilages in serous and synovial vities, the cartilage found in encysted tumors, and in the parenchyma f organs, as the uterus or thyroid gland, and what are called cartilagious transformations of serous membranes and of the inner coat of the essels.

§ 2. *Deviations in Size.*—There appears to be no genuine hypertrophy f cartilages. Not unfrequently, indeed, they are found irregularly wollen in joints, in which fibroid growths hang from the synovial memrane, in which the articular ends of the bones are deformed by gout, c.; but this is a consequence of disease of their texture, especially of eir intercellular substance. The same diseased condition precedes eir wearing down or atrophy, which is often found considerably adnced at the same time that this apparent hypertrophy exists elsewhere. trophy, like hypertrophy of the cartilage of joints, occurs only when there has been some previous disease of their texture: it chiefly affects the large articulations, selecting especially the hip and knee. The loss of substance goes on gradually, and quite independently of contact with purulent matter in the joint.

On opening such a joint one or more spots of various size are found at which some of the substance of the cartilage is lost. The margins of the defective spots are irregular, sinuous, and indented; the depth to which they reach varies, but they very commonly extend through the whole thickness of the cartilage. If any of it still remain, it is of a dull white color, and has lost its glistening and homogeneous appearance; it seems to have assumed a filamentous velvety texture, and is at the same time softer than natural, moist, and succulent. Not unfrequently it is covered over with a cellulo-gelatinous vascular substance. When the cartilage has been removed in its whole thickness, there is some variety in the state of the surface of the bone: it is sometimes, and no doubt always when first denuded, invested with a delicate cellulo-gelatinous organized substance, which fills up the aperture in the cartilage: at other times, especially at a later stage, it is covered with a white fibroid tissue, into which the organized substance just mentioned has been converted; in a third case, it may be literally laid bare, and it then has a smooth appearance, as if it had been polished; it is whiter than the adjoining part of the bone, and denser, as well as whiter than natural, to some depth from the surface.

The cartilages are very frequently found thus changed in some one part of a joint, when no alteration is perceptible in the texture of the other articular structures, or in the character of the synovial fluid. But when the cartilage is lost at several places, and the defective spots are more extensive and coalesce, various anomalies are noticed in the other articular structures,—anomalies which are compounded of those already depicted at pp. 139 and 159, as consecutive induration of the articular extremities of the bones, and a derangement of the joint, which is probably of gouty nature.

Attention has recently been attracted to this disease of articular cartilages, by the interest which the subject of diseases of joints has excited, especially in England, and more particularly with reference to their etiology and nosological import. In my own opinion, which is founded upon repeated investigations, the wasting of the cartilages is occasioned by some previous disorder in the cancellous structure of the articular ends of the bones, especially by their more or less intense inflammatory rarefaction, and consequent condensation. The first derangement of the texture of the cartilage is produced by the exudation effused into it from the adjoining bone. As the secondary process of induration goes on, the nutrient vascular apparatus of the spongy extremity of the bone becomes atrophied; and the further the induration advances, the more certain is the wasting of the previously diseased cartilage, for the extent and the degree of its atrophy correspond with the extent and intensity of the induration. The idea that this loss of substance arises from a true ulcerative destruction is altogether erroneous; neither at the spot itself, nor in the healthy condition of all the other articular structures, is there anything to countenance it.

In advanced cases, the cartilages may be entirely absorbed, or reduced to a few thin remnants, which are commonly situated at the borders of the articular surfaces. The opposite bones are thus brought into contact, and the ivory-like polish of their surfaces makes up, in some measure, for the loss of the cartilages. The disease is commonly known in Germany as the eburnated condition of joint, the name which was given to it by Lobstein. In its higher degrees it is associated with other anomalies, which have already been mentioned as occurring in the articulations, and which have yet to be collected and arranged together in the article on Diseases of Joints. It must be distinguished from other cases in which a similar, but much less marked ivory condensation and polish of the articular surfaces of the bones, succeeds to the destruction of the articular cartilages by ulceration, and in which, accordingly, the change in the spongy tissue of the bone is not the primary affection, but is due to the loss of the cartilage.

Atrophy of cartilage, and the change in the cancellous structure, which gives rise to it, are most frequently met with in advanced life. It is a remarkable fact, to which attention was first directed by Gulliver, that the disease is apt to occur symmetrically in corresponding joints.

The fibro-cartilages, which are most subject to atrophy, are those of the spine. They are found wasted in curvatures of the column, especially in considerable lateral curvatures; and so much of them may be absorbed in the concavities of the curves, that the bodies of the vertebræ come into immediate contact, and unite together. But, under other circumstances, they display a power of resistance, which surpasses that of the bones; and it is remarkable how little they yield to the pressure of aneurismal tumors. Moreover, in old age the intervertebral disks are subject to a form of atrophy, in which, first, the soft cartilaginous substance that enters into their composition is absorbed, and at a later period, their fibrous texture: they become dry and friable, their central gelatinous portion becomes fibrous, and partly disappears, leaving a cavity, which, as well as the peripheral parts of the disk, is filled with fat: the whole structure takes on a dirty yellowish, or rusty brown appearance, the fibres losing their lustre, and becoming dull.

§ 3. *Solutions of Continuity.*—Lacerations of cartilages never occur without great external violence, and they rarely happen alone; either fractures of bone or lacerated wounds of soft parts being combined with them. Fractures of the costal cartilages are the most frequent; and either some ribs are broken at the same time, or the thoracic viscera are ruptured. Lacerations are met with also at the synchondroses of the spine and pelvis; and incomplete separations occasionally take place at some of the pelvic joints during difficult parturition.

These injuries are repaired in various ways. The uniting medium is never found to be new cartilaginous tissue. For the repair of injuries to true cartilages, the inflamed perichondrium supplies an areolar substance, somewhat like ligament, or else a bony callus. The tendency to reunite in the latter way seems to be regulated by the proneness of the cartilage to ossification when in its natural state. Thus fractures of the costal cartilages generally unite by means of a cup, or ring-shaped bony callus, which encloses the fractured part in a sort of capsule. Injuries to the fibro-cartilages heal like wounds of cellular tissue and tendons.

§ 4. *Diseases of Texture.*—The principal disease to which cartilage is liable is,—

1. *Inflammation*, a subject of much discussion, on which its anatomical characters and experiment have been alike brought to bear.

From our present knowledge of the texture of cartilage, as well as from direct observation, we are compelled to deny, that true cartilage, while its texture is normal, ever inflames; but we know by experience that, without being the immediate seat of an inflammatory process, such a cartilage may suffer very serious, and sometimes very rapid, lesions of its texture, from the effect produced on it by inflammation of adjoining tissues, and by the products of that inflammation, and even from imperceptible anomalies in the composition of the synovial fluid. No doubt it is this sensitiveness of cartilaginous tissue to the influence of neighboring inflammation, and inflammatory products, which has led to the presumption, that cartilage does itself inflame; and such a presumption would seem confirmed, if the adjoining inflammation were regarded as secondary to that of the cartilage. The structures which inflame in such close contact and relation with cartilages, and on which the changes in the cartilages now under consideration may in some measure be studied, are the synovial membranes of large joints, the articular ends of bones and perichondrium. The last-mentioned structure may be the seat of a primary inflammation, or may become involved in inflammation or ulceration going on in other tissues, such as mucous and serous membranes, muscles, &c. The vascularity which Mayo and Liston have observed in cartilages during their absorption, and to which I have also referred at p. 212, is without doubt a subordinate occurrence, coming on when the texture of the cartilage is already diseased. When it is in this secondary condition, it may of course become the seat of inflammation, and a free product, consequently, may be found exuded on the surface of parts at which absorption is going on.

In cases of slight inflammation of synovial membrane, and even in those in which, from old age, or from the joint having been unused for some time, the synovial fluid has diminished in quantity, and at the same time probably undergone some change in its constituent elements, the articular cartilages lose the lustre of their surface, without becoming reddened, or perceptibly injected. As they continue longer in contact with the exudation, they assume a yellow color, and a looser and softer texture than natural, so that they look like short-napped felt. On minute examination, the intercellular substance is found at first to be rendered opaque by the presence of a number of fine points and indistinct filaments: afterwards the filamentous arrangement becomes more distinct, and is found to be produced sometimes by fibrils of wavy cellular tissue, and flattened fibres, which are degenerating and assuming a similar character,—both which, as well as the rest of the intercellular substance, swell, and become transparent, when treated with acetic acid,—and sometimes by delicate, nucleated fibres spread out into a membrane.

The cells are round, as if distended, and project from the fibred blastema; in many of them the nucleus is indistinct, and breaks into small rounded points, or degenerates into a fat-globule: in the latter case there is a good deal of free fat in the blastema, and the cells are filled with small shining molecules of fat. When in this state, just as in atrophy, the cartilage is gradually worn down and lost.

When the cavity of a joint is filled with purulent exudation from the synovial membrane, the most superficial layer of the cartilages, being in contact with the matter, loses its glistening and translucid character, and becomes of a dirty yellowish color; but as it gradually changes into a gelatinous substance, it becomes transparent, though it still remains discolored. The proper texture of this layer is completely destroyed, and it degenerates into a finely granular mass. The changes already described go on in the subjacent layer; that is to say, the intercellular substance assumes a fibrous character, and fat is developed in remarkable abundance. When the costal cartilages suppurate in this manner, groups and bundles of peculiar, stiff, straight fibres are sometimes formed in the intercellular substance.

These changes are sometimes found occupying the whole surface of the articular cartilages uniformly, but more commonly they are confined to certain spots of various sizes.

If the articular ends of the bones be acutely inflamed, some of the product of the inflammation which is effused upon their surface loosens the cartilage from them; and in that case the change above described takes place in the cartilage, so soon as the inflammation has extended to the synovial membrane, if it have not done so already.

The same changes in cartilage ensue when its perichondrium is inflamed, and the purulent matter is effused on the inner surface of that membrane: of this we have an instance in inflammation of the investing membrane of the cartilages of the larynx. They suffer in the same manner if the perichondrium be destroyed by ulceration advancing from other organs; as is the case in the cartilages of the larynx when situated at the base of degenerated typhous, or tubercular ulcers; or in the cartilages of the ribs, when there is any tuberculous ulceration of the costal pleura.

It is an old and interesting observation, that ossification is apt to be induced in cartilages by the occurrence of inflammation in their vicinity; but it takes place only in those cartilages which by their physiological constitution are wont to ossify, such as the cartilages of the ribs and larynx.

Inflammation, though rarely met with in the fibro-cartilages, does unquestionably occur in them; but the changes it produces in them differ somewhat from those just described. It is remarkable for its acute course, and for the rapid ulcerative destruction of the fibro-cartilage to which it leads: its anatomical characters are much the same as those of inflammation of fibrous organs. An inflammation is sometimes met with in the intervertebral cartilages, which terminates sooner or later in suppuration, and is generally in the end combined with inflammation and caries of the bodies of the vertebræ, inflammation of the spinal membranes, &c.

After difficult parturition, the synchondroses of the pelvis, and especially the fibro-cartilage of the symphysis pubis, are subject to an inflammation of low type, which owes its origin to putrefactive (septic) phlebitis, and rapidly destroys the fibro-cartilage in a similar manner to the worst forms of putrefaction. (Compare p. 55.) The cartilage is found enclosed in a capsule formed of the adjoining ligamentous apparatus, but it is changed into a diffluent, fetid pulp, resembling chocolate or coffee-grounds, or into a fluid sanies. Sometimes the disorganized mass is found in an irregular cavity, having insinuated itself to various distances beneath the periosteum of the neighboring bone.

In the cartilages which Miescher has named the yellow cartilages, and which have a general connection with those now under consideration, an inflammation occurs, which is mostly chronic in its course, and which, after loosening and swelling the texture of the cartilage, usually terminates in atrophy and fibroid induration of it, and consequently in deformities of various kinds. Hence the epiglottis, and more frequently some of these structures about the tarsus, are found puckered, bent, tilted upward or downward, &c.

2. *Ossification of cartilage.*—Some of the true cartilages are, under various circumstances, subject to real ossification, to conversion into genuine bone. The thyroid and cricoid cartilages ossify in this manner in men at about forty years of age: and ossification of the costal cartilages is so frequently observed in old persons that it can only be looked upon as a pathological appearance when it is found at an earlier period of life. The cartilages of the larynx ossify from different points: those of the ribs change to a dirty yellow color in old age, become saturated with fat, and then ossify in the middle, the central vascular canal enlarging so as to form a medullary cavity. The ossification of those laryngeal cartilages which I have mentioned, is often excited and advanced by inflammations which extend to the perichondrium from without, especially the inflammation which occurs at the base of tubercular ulcers and in the neighborhood of softening tubercle. Hence it sometimes happens that tracheal phthisis is after a time complicated with caries of the newly formed bone.

True ossification of a fibro-cartilage is in every case highly problematical. The anchylosis of synchondrosis which appears to result from such a change in the fibro-cartilage, is probably never produced by conversion of that structure into bone. Either new bone, in the form of an osteophyte, passes between the margins of the adjacent surfaces of the anchylosed bones, enclosing the fibro-cartilage in a sort of capsule; or the two bones are united together in the whole extent of their adjoining surfaces: in the latter case, there can be no doubt that the new osseous matter is formed on the surfaces of the old bone, while the fibro-cartilage is absorbed. In this class of cases are included those rare ones of complete anchylosis of the synchondroses of the pelvis and of the vertebral bodies; and allied to it is another kind, in which the vertebræ are fused together on the concavity of lateral curvatures in consequence of the absorption and displacement of the intervening cartilages by pressure.

n the other hand, several of the so-called yellow cartilages are in rare cases, the seat of a bony concretion, which is formed in some id callus left in them after chronic inflammation. The epiglottis is nstance of this.

. *Adventitious growths.*—These growths appear not to occur in the ilaginous system, and certainly they do not in true cartilage. Its cture will resist the progress of advancing cancer for a long time, even altogether. The costal cartilages do indeed disappear in the eneration of large mammary cancers, and the arytænoid cartilages a very remarkable exception to the rule; for from what I have erto been able to ascertain, it appears as if the cancerous growths, hose suspected to be cancerous, which project into the larynx, very monly spring from those cartilages. Tuberculous and cancerous nerations affect the cartilages in the manner already described under head of Inflammation.

APPENDIX.

ANOMALIES AND DISEASES OF JOINTS.

all deviations from the healthy condition of Joints, the diseases of ure are the most serious, both on account of their frequency, of r relations to other diseases, and of the varieties which they present heir earlier stages, according to the particular structure that was ted first, among the many of which the compound apparatus of ts is composed. Moreover, they very often lead to alterations in the tive position of the bones, and in the form of the joints.

1. *Deficiency and Excess of Development.*—Just as, when one or e bones are wanting, there is a deficiency of the corresponding ts, so in the opposite case, supernumerary bones are articulated to skeleton by additional joints. But sometimes joints are really nt; or, when the bones which should compose them are incompletely loped, a mere indication only of the articular structures exists. osseous framework is partially developed and rudimentary; the ule of the joint is very small; and when the bones fail much of r complete development, though the stump which represents their ular extremity be covered with cartilage, the capsule is detached, the ligaments are partly or altogether wanting. The joints by h supernumerary bones are attached are very commonly defective; in congenital anchylosis the articular apparatus is entirely absent.

n the contrary, supernumerary joints are met with, not only when e are more bones than usual, but when their number is natural. an unfrequent example of the latter fact is shown in the connection djoining ribs together by means of an articulation.

§ 2. *Deviations of Form.*—These deviations are extremely numerous and very varied in their character: sometimes they are congenital, and are caused by some fault of original formation; at other times, and far more frequently, they are secondary, and occur either before or after birth, as consequences of permanent dislocation of the bony structures of the joints, or of disease of their texture.

The cases of the former, or congenital, class may in general be reduced to unnaturally large or small size of the heads, or prominences in joints, on the one hand, and to increased or diminished capacity of the fossæ on the other. And the shape of articulations may be altered in various ways by the shortness or unnatural position of articular processes, or by peculiarities in their attachment to the shaft of the bone; a fact of which the import has been shown in the instance of the neck of the femur.

The deformities of joints which are acquired after birth, are much more numerous and more important: they consist, in general, of some uneven enlargement, flattening, or diminution, or of complete destruction of the head of an articulation; of dilatation, narrowing, or wasting of articular cavities; of bending of bones, and their articular processes, &c.

§ 3. *Alterations in the Contiguity of the Structures of Joints.*—The chief of these anomalies are dislocation and anchylosis.

Dislocation consists in the slipping of the portions of bones which compose a joint over one another, so that they enter into new relations of contiguity. It is evident that there may be many degrees of it; and hence a distinction is drawn between complete luxation and incomplete, according as the articular surfaces are entirely separated, or still remain in some contact with each other.

There is a difference, too, between luxation from violence and spontaneous luxation, which occurs in the course, and is a consequence, of inflammation and ulcerative destruction of joints, but yet, as will appear, is a consecutive, and not an essential, phenomenon of the disease. The former is a result of violent injury to a joint, or of transient or persisting immoderate muscular action, and it occurs most frequently in joints which have freedom of motion. The greater the displacement, so much the more is it complicated with stretching, or rupture of the ligaments of the joint, and even with laceration of muscles, vessels, &c. Rupture of the ligaments most commonly occurs in the dislocation of joints which have limited motion. Unnatural width or shallowness of the cavities of joints, and too great length and laxity of their ligaments, predispose of course to dislocation, and to the occurrence of it upon slight occasions. The facility with which dislocation occurs in some individuals, and their ability to produce it at will (habitual dislocation), depend upon the same physical condition.

Spontaneous luxation is usually the result of destruction of the bony parts of a joint, and of the reaction of the surrounding muscles which takes places in the course of the disease: the extent of displacement corresponds with the amount of destruction which has occurred. A further account of this condition will be more conveniently given below.

Lastly, luxation may be either congenital or acquired. Congenital luxation has been only lately recognized: it has been observed in several joints, chiefly in the hip. Guérin regards it as probably the consequence of muscular retraction in the fœtus, just like club-foot, which is essentially a dislocation. Various other dislocations may, like club-foot, be produced at later periods of life by muscular retraction. Congenital luxation, when examined in a grown-up person, does not present such striking characters that it can be distinguished with certainty from a dislocation which occurred in early childhood, or even from one produced by muscular action before the bones were completely formed. It appears to me to require a thorough examination of the patient in order to determine any particular luxation to have been congenital; for, in the hip-joint more particularly, the changes which are produced by coxalgia bear a deceptive resemblance to congenital luxation, and many cases of that disease have, in the preparation, been looked upon as congenital dislocation.

Luxation is followed by some more remote changes in the joint. The capsule becomes enlarged, and the place of its insertion altered; the articular cavities of the bones increase in size, and undergo various changes in form; and corresponding alterations are produced in the articular heads or prominences. In other cases, in which the dislocation is complete, the capsule wastes, and the bony cavities diminish in size, or are filled with masses of new osseous substance; the displaced head of the bone loses its character, and a new joint is formed. The cellular structures which surround the dislocated head inflame, and frame a new capsule around it, which, for the most part, fits closely, is of fibroid structure, and has a serous lining; whilst the pressure of the head in its new position occasions a shallow articular excavation beneath it. In other cases, instead of an excavation beneath the head, a mass of callus springs up around it, and forms either a hollow to receive it, or a level surface, which the head may be flattened in order to fit; or, lastly, the callus may project, and that which was the articular head be excavated to receive it. Sometimes the quantity of new bone deposited around a dislocated head is very abundant, and retains it firmly in its place.

In dislocations of long standing, the pressure upon the vessels and nerves interferes with the nutrition of the luxated bone, and, like the soft parts, it is found in a state of atrophy.

Of *anchylosis* (stiffness of a joint) there are many degrees. In the highest the joint is fixed by means of bone,—two bones which were connected by articulation, unite into one—synostosis. In slighter degrees the synovial membrane wastes, the cavity of the joint is obliterated, the articular extremities of the bones become bound together by means of a fibroid or cellular tissue, and the joint is still capable of some motion. These constitute true anchylosis, and must be distinguished from the stiffness of a joint which is produced by swelling, shrinking, and tightness of the fibrous ligaments, or of the fasciæ which surround a joint, or that which arises from a contracted state of the muscles, &c. To such the name of false anchylosis is commonly applied.

Anchylosis is, in most cases, the consequence of well-marked inflammation of a joint, and frequently occurs, therefore, after injuries in its neighborhood, or when caries or necrosis encroaches upon it. But it comes on also without causes of this kind in joints which have long been unused; especially in contracted joints, the articular surfaces of which have remained for a long time in close contact with one another; and in old age, in which the diminished secretion of synovia probably gives the first occasion to structural disease, and ulceration of the cartilages of the joint.

In bony anchylosis the articular ends are either bound together by flattened or rounded bridge-like growths of bone which frequently follow the course of the fibrous ligaments; or they are so united in their whole thickness, that the two or more bones which composed the joint now form but one. Sometimes, on making a section of an anchylosed joint, in which there has been no previous loss of substance in the bone, a streak is seen which represents the compact articular surface; but if that have been destroyed by suppuration, the spongy substance of one bone is uninterruptedly continuous with that of the other.

Anchylosis is sometimes confined to a single joint, sometimes it exists in several: a few cases have been observed in which nearly all the joints were anchylosed, and most of the synchondroses also.

The joints in which anchylosis most frequently occurs are the hip, the elbow, and the knee; next in order to them are the joints of the foot and hand, then the shoulder, and the articulation of the atlas and occiput, whilst that of the lower jaw, and the sterno-clavicular, and acromial joints are very rarely affected.

§ 4. *Solutions of Continuity.*—To this class belong fractures of bone within the capsule of a joint, lacerations of the capsule and ligaments, disruption of the interarticular cartilages, of the cartilaginous coverings of a joint, &c., and all of them are results of violence.

§ 5. *Diseases of Texture in Joints.*—These diseases commence either in the fibro-serous capsules, or in the bones, and scarcely ever in the cartilaginous coverings of the ends of the bones or in the interarticular cartilages. In the course of disease, however, all the different textures composing the joint are affected, and in different ways: this remark applies especially to inflammation, which is by far the most frequent disease of joints. In treating of it, therefore, as well as of the other diseases of texture to which reference must be made, I shall observe that distinction in a manner as closely accordant with nature as possible.

1. *Inflammations.*

a. Inflammation of the synovial membranes of joints.—In the consideration of this, as well as of the other diseases of synovial membranes, frequent reference must be made to the statements contained in the chapter on the Diseases of Serous Membranes in general (p. 27).

Inflammation of a synovial capsule is sometimes a primary disease, and occasioned either by various injuries, such as fracture within or near the capsule, dislocation, &c., or by atmospheric influences; in the latter case it is named rheumatic: sometimes it is secondary, and results from metastasis. It is also very frequently brought on by the inflammation of an adjoining tissue, as of the bones, or their articular extremities, or by caries, necrosis, &c., in the neighborhood of the joint.

Secondary inflammations—those which are the result of metastasis—occur chiefly in the larger joints, and especially in the knee.

Moreover, the inflammation may have an acute course, or may be chronic. In either case, it may produce any of the different exudations mentioned in the chapter that has been cited, and may have in general the same terminations. The best-marked inflammations in the structure under consideration are those which furnish a purulent or a hemorrhagic exudation, and those the product of which becomes converted into tubercle. When the amount of exudation, and the consequent enlargement of the synovial membrane, are considerable, and when the disease involves, on the one hand, the cellular, muscular, and fibrous soft parts, and the ends of the bones on the other, the whole disease constitutes that affection which, from certain stages of its progress or from some one prominent character, is commonly called white swelling (fungus articulorum, spontaneous luxation, Arthrocace). A case of this kind is most marked when the inflammation produces a purulent exudation, and terminates in suppurative destruction of the tissues of the joint, and when the exudation is tuberculous. Commencing originally in the synovial membrane or in the articular ends of the bones, this affection generally, when it is far advanced, involves such various textures, and produces such degeneration and destruction of them, that it costs some trouble to determine its original seat. And it is further important to remark, that whilst it is not altogether uncommon for it to commence in the articular ends of the bones, yet the synovial membrane is far more commonly the organ first diseased. The inflammation of that membrane very often occurs in several joints at the same time; and with chronic inflammations of the larger synovial membranes, there frequently coexist inflammations of the same character, so far as the products are concerned, in large serous sacs, such as the pleura, the pericardium, &c.

A second, and yet more important observation is, that no exudation is deposited on that portion of the synovial membrane which covers the cartilages; if any of it should adhere to that portion of the membrane, it is in no case, whatever form it assume, to be regarded as a primary peripheral plastic effusion, but as a secondary precipitate, or deposition out of the general exudation in the articular cavity; and the opacity of the cartilages and of the layer of synovial membrane covering them is the result of imbibition.

Slight acute inflammations produce rather a turbid exudation, which is for the most part serous, and is easily reabsorbed. They terminate in resolution.

If the product of the inflammation consist of an effusion of serum and of a larger quantity of plastic material, the synovial membrane loses its glistening aspect, becomes opaque and more or less swollen, and is covered on its inner surface with a peripheral plastic effusion, which varies in thickness, and in the character of its free surface. Sometimes it constitutes a thin layer, sometimes a thick one, of uniform or of unequal depth: and as in the latter case there are some extensive spots on which it is scarcely perceptible, and other smaller ones, on the contrary, at which it is accumulated, it forms islands or "plaques" of various, and sometimes very considerable thickness, or even nodulated masses. When it is of trifling thickness, its free surface usually appears fine and villous, but when its thickness is considerable, the surface is shreddy. Very frequently also there is found cleaving to the surface more or less of a soft precipitate out of the effusion, which can be easily removed.

As the whole process is usually chronic from the first, or becomes so in its course, the effusion which remains, and the enlargement of the capsule, render any adhesion and union of the surfaces very rare; and the plastic effusions are converted into a new tissue, composed of free or of coarse shreds, and of fibrous (fibroid, fibro-cellular) texture, which adheres to the synovial membrane.

Specimens are to be met with in nearly every collection, of large joints (especially knee-joints) in which the capsule is enlarged and thickened, and its inner surface covered with white shreds of various lengths, which are occasionally so numerous, that the joint seems as if it were lined with felt; sometimes the shreds are simple, smooth, rounded, or rather flattened threads, or they here and there form a membranous patch upon the surface, or have their free extremities split into filaments and resembling a tassel. In extreme cases, small, smooth, and subovate bodies, which Mayo compares to melon-seeds, are attached to their extremities, hanging singly or in clusters from each stalk: and lastly, here and there amongst them shapeless masses are attached by broader bases. They all have a fibrous or fibroid texture, and are of innocent character, having nothing in common with the cystic and cancerous productions which are found on normal or anomalous serous membranes.

The cartilages of the joint and the bones remain, in many cases, uninjured throughout the process: and excepting a perceptible turbidity, nothing unnatural is found in the synovia. In other cases, on the contrary, the cartilaginous coverings of the articular ends of the bones become diseased. Such cases are commonly looked upon as rheumatic affections of the joint; but chemical analyses of the original exudation, and of other secretions and excretions in the course of the disease; are required to establish their true nature. The cartilages become swollen, and thicker than natural, and usually uneven; they acquire a marked opacity, and dull white appearance, and gradually assume a fibrous texture. If the disease do not materially interfere with the use of the joint, the cartilages, when thus changed in their texture, and the menisci, or interarticular cartilages, are gradually worn away: and the exposed surfaces of the bones continually rubbing against one another, become dense and polished like ivory. The synovial membrane in such joints becomes still more turbid, of a dirty grayish color, and even flocculent. The progress of the disease is very slow, and frequently is quite unnoticed; indeed many observers have on that account been led to doubt its inflammatory nature.

In yet another class of cases the articular extremities of the bones are diseased; and they may be either primarily affected, or secondarily, by the advance of disease to them from the synovial membrane. I shall have occasion to describe these cases among the inflammations of the articular ends of the bones, as a change in joints probably dependent on gout.

Inflammations with a purulent, and those which have a sanious exuda- on, are on several accounts serious; because they frequently terminate suppuration (phthisis) of the various tissues of the joint; because of e extension of suppuration beyond them; because of the so-called spon- neous dislocations of the bones which ensue; and, lastly, because of e anchylosis in which they terminate in favorable cases.

The quantity of purulent fluid effused into the cavity of the joint is nerally considerable, and the capsule is consequently much enlarged: e synovial membrane is lined with a firm, shreddy layer of lymph, hich is dissolving into pus, and a softer purulent precipitate, which can easily removed, adheres to the cartilages. The first-mentioned layer opaque and lustreless, its surface is rough, and serum is infiltrated, d blood in small spots extravasated through its tissue, as well as rough that of the fibrous capsule of the joint, and neighboring cellular ructures. As the disease advances, the infiltration and thickening of e neighboring structures increase, they become filled with a gelatinous, rdaceous, white product, in the midst of which fibrous tissues, capsule, gaments, or aponeuroses, can no longer be recognized. Here and there, the mass, there are cavities of different dimensions, the lining of which vascular, spongy, and granulating, and the contents purulent. The uscles near the joint are pale and flabby, infiltered and attenuated. t length the infiltration reaches the subcutaneous cellular and adipose ssues, and the integuments become fixed to the disorganized structures neath. The diseased joint then presents the following external ap- earance: it is swollen, and always more or less bent; it feels everywhere ft and flabby, or in some spots flabby, in others firm, elastic, doughy, d, at the same time, tuberculated; the integuments over it are tense d pallid,—leucophlegmatic, or they are traversed by varicose veins. White swelling. Fungus articuli.)

At length ulceration commences, and advances in various directions. xternally, the capsule ulcerates in one or more spots, and then the soft arts adjoining it. In some instances, large openings form in the cap- ule, and connect the joint with ulcerated cavities in the soft parts; in thers, mere sinuses are formed: but in either case they open externally rough the skin, and occasion and maintain a discharge of the contents the joint. Internally, the interarticular cartilages and the ligaments lcerate, the cartilage covering the bones, when brought into contact ith the matter, is destroyed in the way that has been mentioned, and e ulcerative inflammation attacks even the bones, if they have not een involved already.

The cavity of the joint appears like a cloaca, surrounded with a gela- no-lardaceous mass: the integuments covering it are of a dark-red hue d are especially discolored at the orifice of the sinuses. The joint ntains pus or sanies of an offensive odor, and variously discolored, the peated hemorrhages which take place when there is acute caries of the ones, very frequently giving it a red or brown tinge: the ligaments lcerate, and the cartilages separate, partly or entirely, from the bones: e osseous surfaces are laid bare, their compact wall is destroyed, and e spongy tissue is exposed, infiltered with pus and ulcerating, and sur- unded on all sides by osteophytes of various shapes; remains of the fibrous structures of the joint, pieces of loosened cartilage, and of necrosed bone, are mixed with the matter discharged from the joint. The soft parts, and the entire bones belonging to the diseased joint are wasted, most of the fat is absorbed, the muscles are remarkably blanched and thin, and the bones, being generally in a state of excentric atrophy, are soft and fragile.

More or less quickly after the disease has reached this stage, spontaneous dislocations, as they are called, ensue. In modern times doubt has been cast upon the fact of dislocation occurring in consequence of large effusion into a joint, but experience certainly proves that such dislocations do take place. The head of the femur has, in some few cases, been forced out of the acetabulum by a large exudation into the hip-joint; and thus the views of ancient authors on the subject of Coxarthrocace, though, in many respects, unquestionably defective, are justified, and the manifold criticisms to which they have been subjected, are corrected.

The greater majority of these luxations occur in the last-described stage of an inflammation attended with purulent exudation, and are consequences of the destruction of the ligaments and articular ends of the bones; they are brought about by the continued action, and follow chiefly the direction, of the flexor muscles; though if the bone be much destroyed towards any other part, the direction of the dislocation will be modified accordingly.

The most favorable case of repair of this disease, is discharge of the matter, and anchylosis of the joint. The cavity of the articulation is obliterated, being filled with a new growth of organized cellular tissue, and with an abundance of fat. The bones become knit together, either by means of an ossifying exudation, which is furnished by their compact articular surface, or, if caries have taken place, by the granulations which spring from their exposed spongy tissue; and the more closely the bones are in contact, the earlier is the union completed. The soft parts around, which have been inflamed, infiltrated and ulcerated, contract, assume a white fibroid texture, and cicatrize; and the whole joint appears wasted.

Inflammations, which furnish a hemorrhagic product, take place under the general and local conditions which are peculiar to them: they chiefly attack large joints, and especially the synovial membrane of the knee. They are usually associated with hemorrhagic effusions into large serous sacs.

In those inflammations, the product of which becomes converted into tubercle, the same conditions may be observed, which have been adduced as applying to inflammations of this kind in serous membranes in general; they constitute the usual, and almost the only form in which tuberculosis occurs in synovial membranes. They lead to tubercular ulceration and destruction of the tissues of the joint, and are the cause of a great number of what are called "white swellings." I shall have to advert to this subject again.

b. Inflammation of the spongy articular extremities of bone.—Inflammation, and the inflammatory osteoporoses or rarefactions, of bone having been already considered, there are only a few special remarks to be made in this place.

The articular ends of bones are frequently the seat of inflammation, especially in young persons; and the inflammation often passes into caries. Inflammation of the synovial membrane, of course, accompanies it, with equal readiness and frequency; and there is great difficulty in any particular case of inflammatory disease, especially in one of ulcerative destruction of a joint, to determine in which structure the disease originated.

As a general rule, that structure was the first diseased, in which the inflammation and destructive process are most advanced. If there be a considerable production of new osseous substance (osteophyte) around the articular ends of the bones, and on those portions of their articulating surface which have been divested of cartilage, if the ends of the bones be much enlarged, and their tissue expanded, if they be extensively carious, if matter be formed in their interior, but especially if the cartilage covering them be loosened, and the epiphyses separated, it is highly probable that the original disease was in the bones. The inflammation of the bones not unfrequently leads to an exudation which bears the characters of tubercular matter,—to a tubercular infiltration, in fact, of the spongy tissue of the bone; when this occurs, it is usually under the influence of a deep tuberculous taint of the system, which has, moreover, already exhibited itself by a tubercular deposition in some other part. Inflammation of the synovial membrane, attended with an exudation of tubercular matter, generally soon follows. (*Vide* Tuberculosis of Joints, *infrà*.) The disease next in order is that form of—

Osteoporosis of the articular ends of bones, which I have, at page139, distinguished as inflammatory; it ends in a particular kind of induration of the osseous tissue, and produces peculiar deformities of the bony apparatus of joints. An attempt to delineate it was made (p. 159) in the chapter on the characteristic features of Constitutional Affections of Bone, where it was represented as a change very probably dependent on gout.

The disease of the joint commences with loosening or expansion of the tissue of the bones and enlargement of their articular ends, and it evidently is chiefly confined to the prominences or articular heads. This softening and swelling of themselves occasion various deformities of the bony structures of the joint, and they are usually rendered still more striking by exuberant growths, which spring up simultaneously around the articulating surfaces. The most common deformity is that in which articular prominences become flattened, and their margin overhangs to such an extent, that their whole form resembles that of a mushroom: a corresponding widening and flattening take place in articular fossæ. But an articular prominence, especially the head of the femur, may be deformed in another way: it may maintain its shape, but still have an overhanging border; or it may be flattened from above only, and, at the same time, depressed; and may have a groove or impression at its upper circumference: in other cases it is elongated and conical; in others, again, uneven and tuberculated. Corresponding to each of these deformities of articular prominences, there is a deformity of the fossæ which they occupy. The expansion of the bony tissue (the osteoporosis) gradually passes into a white chalky condensation, or sclerosis.

The cartilages of the joint are gradually removed, and their removal is effected by several means. In the first place, their texture is probably altered by an exudation, which is poured out during the process of rarefaction upon the articulating surface of the bone. As the exudation thoroughly saturates the cartilages, their blastema assumes, as has been remarked, a fibrous structure, and they become of a dull white color, and, at the same time, softer and more moist. This change of texture occurs the sooner if the disease in the bone be accompanied by inflammation of the synovial membrane, and effusion into the cavity of the joint. Whilst the cartilages are thus prepared for removal, the condensation of the bone, and the rubbing of the altered cartilages themselves against each other, effect it. The one impedes or prevents their nutrition by the vascular apparatus of the spongy tissue of the bone, the other wears them down; and does so with a rapidity proportioned to the loss of elasticity and of resisting power, which the change in their texture has occasioned.

When the cartilaginous coverings are removed, the continued attrition gives the condensed articular ends of the bones a polish like that of gypsum. At the same time, the interarticular cartilages, which have become useless from the deformity of the articulating surfaces, and have suffered more or less compression, are also worn down and removed. And in the hip-joint, the ligamentum teres always disappears, partly because it is wasted in consequence of the induration of the bone, and partly by being worn away.

The inflammatory process extends from the bones to the synovial membrane. The capsule is almost always thickened; internally it is frequently found of a rusty brown or slate-gray color, and lined with a false membrane, that has a delicate villous surface, or with the fibroid growths before mentioned. The fluid in the joint is thin, turbid, and of a dirty grayish color. Moreover, the capsule of the joint is dilated in proportion as the articular fossæ are enlarged; and its insertions extend beyond the normal bounds of the joint.

For other anomalies of the same kind, as well as for the enumeration of the joints most frequently affected with them, reference may be made to what has been said at other parts of this work; there is but one remark to be added here, which is, that the disease not unfrequently occurs symmetrically in corresponding joints.

2. *Adventitious growths.*

a. Lipoma.—This occurs in joints under the form which Johann Müller has distinguished by the name of lipoma arborescens,—a branching growth of fatty tissue in the free part, or in the duplicatures of synovial membranes; it occurs chiefly in the knee-joint.

b. Fibroid tissue.

a. When it occurs in connection with the synovial membrane, it is formed in old exudations; and it adheres to that membrane, assuming some of the manifold shapes already mentioned (p. 175). In some of those forms it accumulates in great abundance. All the loose bodies (or mice, as they are called), which are met with in joints, or at least a great part of them, belong to this class. (Comp. p. 43). They are either masses of exudation set free from the inner surface of the synovial membrane, or growths which have originated beneath it, or, lastly, formations (precipitates) from a synovial fluid of unnatural composition. In the last case, they are distinguished by their laminated structure.

β. Fibroid tissue occurs in the articular ends of the bones in the form of fibroid tumors, which sometimes attain very considerable size.

c. *Tubercle.*

a. Synovial membranes are the seat of tubercle; and, as has been casually remarked already, almost its only form is that in which it is a product of inflammation. It then usually coexists with similar inflammations in large serous membranes, and as both are dependent on advanced tuberculosis of parenchymatous organs, especially of the lungs, it is associated also with a great degree of general tubercular disease. It affects chiefly the larger synovial sacs, such as those of the knee, hip, and elbow. When the exudation softens, and suppuration takes place, the capsule of the joint ulcerates; and secondary tuberculosis, and tubercular ulceration of the bones ensue.

The articular ends of the bones are affected with tubercle sometimes simultaneously with the synovial membrane, and sometimes before it.

β. When tubercle is formed in the articular ends of the bones, it is usually a consequence of inflammation, and is diffused through the spongy tissue of the bone. It gives rise to caries, i. e. to tubercular abscesses, which very often break into the cavity of the joint (Nelaton). It is commonly very soon followed by inflammation of the synovial membrane, and tubercular exudation.

Both occasion the most terrible destruction of the tissues of the joint.

d. *Cancers.*

These diseases commence in the articular ends of the bones. The most frequent is medullary cancer, which, after reaching a considerable size, may advance into the cavity of the joint, but never commences, as a primary disease, within or upon the synovial membrane.

§ 6. *Anomalies in the Contents of Synovial Cavities.*—Most of these may be gathered from what has been said already; and there needs only to mention here,—

a. Dropsy of the synovial membrane, which arises from the same causes as dropsy of serous sacs in general. In most cases it originates in a slight inflammatory process, the serous product of which contains very little plastic material.

b. The loose bodies,—articular mice,—which have been already described.

PART XI.

ANOMALIES AND DISEASES OF THE MUSCULAR SYSTEM.

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§ 1. *Deficiency and Excess of Development.*—In very incompletely-formed monsters no muscles whatever, or merely a few traces of them, are to be found. Not unfrequently, when that portion is imperfectly developed, a few, or the whole, of the muscles of some portion of the body are wanting. This is the case with the thoracic and abdominal muscles, and those of the back, when the thorax, abdomen, or spine is fissured; or with the muscles of a limb, when the development of its skeleton is arrested; and some supernumerary limbs, which do not pass beyond a rudimentary state, never have muscles formed in them. A few rare instances occur in which most of the muscles of a limb are wanting, although its skeleton is perfect. And now and then a few unimportant muscles, or parts of muscles of the face, trunk, or limbs are missing, but the want of them does not interfere with the power of motion in the part.

Allied to these instances of deficient development, in which one or more muscles are entirely absent, there are others in which a muscle may be imperfectly developed in bulk (thickness and power), and in texture. Sometimes one of its extremities is but partially formed, or not at all: or it may have no insertion, as is the case with muscles and their tendons at the stump of rudimentary limbs, or when, in the absence of the lower jaw, the muscles of the cheek are fused together with those of mastication. And, lastly, it may happen, that whilst the bony part of a limb is properly formed, the muscles are too short.

The form in which an excess of development presents itself is that of an increased number of muscles. Sometimes unusual ones exist, at other times a muscle has an irregular or accessory head, or an additional tendon; and, occasionally, particular parts are stronger than usual, and are separated by unnaturally deep fissures into independent muscles. In the same class may be included cases in which muscles and their tendons exceed the natural length.

§ 2. *Deviations of Muscles from their natural condition in respect to Size or Volume, and Form.*—Some of the examples of originally faulty development which have been mentioned in the last section, belong to the present also; but there are other anomalies which, though sometimes congenital, more frequently occur after birth, and are, therefore, more properly arranged in this section: the anomalies in question are those of shortening, atrophy, and hypertrophy of muscle.

In *shortening* (retractio, contractura) the muscular fibres are permanently contracted: the cause of their contracting is excessive innervation, and the consequence is gradual wasting of the muscle. It leads to a proportionate deformity of the skeleton of the part, which becomes permanently straightened or bent, and has its articulations partially or completely dislocated, and the bones themselves curved and bent. Whether it be congenital or come on after birth, it is a result of repeated tonic cramp. It is seen in wry neck, in club-foot and hand, in several of the curvatures of the spine, &c.

The circumstances under which *atrophy* of muscles takes place are various; sometimes it is a simple wasting, at other times a complicated. The former kind is distinguished by the muscular structure being paler than natural and easily torn, and is general throughout the whole muscular system; it is a consequence of advanced age, and of various diseases, and is found with gelatinous softening of the stomach and intestines in children, in the phthises, especially tubercular phthisis, in cases of extensive cancerous growth and cancerous degeneration, and in consequence of slow poisoning, especially with lead. An atrophy of the same kind ensues, but is more partial in its extent, when particular groups of muscles have been insufficiently used, and in paralysis. In some muscles it is brought on by the gradual pressure and distension which they suffer from the enlargement of organs, the dilatation of cavities, or from morbid growths: as by an enlarged thyroid gland, dropsical distension of the cavity of the peritoneum, aneurisms, sarcoma, cancers, &c.

A complicated atrophy of muscle is that in which the texture of the fibre is changed. Contracted muscles waste in this manner: they lose their natural color, and become first of a pale yellowish-red, then of a fawn, and lastly, of a dirty white color; while at the same time they degenerate into a firm, tense, fibrous (fibroid) cord. Muscles which are absolutely unused, such as those of anchylosed joints, waste and degenerate into fat. Moreover, the muscles are generally found atrophied when much fat is formed in the body.

A remarkable form of atrophy has been described by Mayo, which in two cases that came under his care, was brought on by long-continued exposure to cold, was attended with much pain, and ran a very acute course. It is a subject on which further investigation is still required; and in order to direct the attention of medical men and pathologists to it, I introduce here the second of Mayo's two cases.

A laborer, æt. 45, who had been much exposed to wet, and was in the habit of allowing his clothes to dry upon him, was attacked, four months before he was seen, with pain in the left shoulder, which continued almost uninterruptedly for six weeks. It was most severe about fourteen days after it commenced, and still became sometimes so violent, that it seemed, he said, as if his arm would drop off. There was neither swelling nor redness; nor was there any numbness or tension when the shoulder was pressed, but he felt great pain when he raised the arm with the other hand. Without that assistance, he could not lift it at all. About a week after the affection had begun, he noticed that the shoulder was wasting. As the pain subsided, the wasting gradually advanced, and at the time when he was examined, the deltoid, supra-spinatus, infra-spinatus, and the two teres muscles appeared to be completely absorbed, or educed to thin layers of membrane. The shoulder was free from pain nd the joint healthy, but yet he was unable to lift the arm. There was o emaciation of the forearm or hand.

The muscular coat of the alimentary tube, and the bladder, are in ome rare cases manifestly attenuated. Their walls become thin, trans- arent, withered, and pale. Sometimes an atrophied condition of the uscular coat of the alimentary tube is met with, analogous to that hich is combined with the growth of fat; the muscular coat of the owel becoming thin, at the same time that fat accumulates in the esentery, the omentum, and throughout the system.

Hypertrophy in the system of voluntary muscles, to an extent that ould be called morbid, and would essentially disturb the functions of he part, is extremely rare, the tongue and a few of the respiratory uscles alone excepted. But it is very frequently observed in the heart, nd in the muscular coat of the alimentary tube and bladder. The degree hich hypertrophy attains in these organs is often exceedingly great. t is mostly due to some mechanical obstruction, to increased action and verstraining of the organ, to immoderate innervation, or, as in the in- tance of the bladder, to catarrh of the mucous membrane. After having eached a certain degree, it ends in palsy of the organ, and in this man- er may destroy life of itself, and suddenly, or it may lead to various onsequences, such as ileus in the intestinal canal, and inflammation and loughing in the bladder, which in the end are fatal.

The deformities have partly been mentioned amongst the faults of evelopment; the remainder are merely those which are produced by etraction, atrophy, and hypertrophy.

§ 3. *Variations in Color.*—The color of muscles varies much under ifferent circumstances. Thus, as will appear in the latter parts of this hapter, diseases of their texture are attended by changes in their color. hen there is general emaciation, in dropsical subjects, in cachectic ases, such as rickets, tubercle, and chlorosis, after hemorrhages, &c., and n paralyzed limbs, the muscles are paler than natural: in advanced life hey have a pale rusty brown, or a dun color. Muscles which waste in onsequence of permanent spasmodic contraction, have a grayish or yel- owish-red hue, and at length become dirty white; when the atrophy is he result of steatosis, they are yellowish-red; and so on. On the other and, hypertrophied muscles are of a deep-red color; and those of per- ons who have died of typhus, acute convulsions, Asiatic cholera, or ydrophobia, of patients with scurvy and cyanosis, and of persons suf- ocated, are of a dark purple-red color, with a shade of blue or violet. uscles in the neighborhood of collections of pus or sanies become rownish-green, or greenish-blue.

§ 4. *Deviations of Consistence.*—There is usually some connection etween the deviations of color and those of consistence: when muscles ose or change color, they mostly become less firm; and when they have deep-red color, their consistence is increased. Muscles thus altered ay be in an unnaturally lax, supple, soft, and friable condition; or they ay be compact and tough; or, again, though firm and glistening, they may be dry and easily broken. Thus, in the forms of cachexia just mentioned, the muscles are not only pale but lax; in old age and steatosis they are discolored, and soft, they tear very easily, and may be squeezed into a pulp; whilst on the contrary, spasmodically contracted muscles are tough, notwithstanding their loss of color. Hypertrophied muscles are firm and strong, and so are those of patients who have died of acute convulsions or hydrophobia, and especially after Asiatic cholera: in the last-mentioned cases they are also remarkably dry.

A still more marked instance of diminution of the consistence of muscles occurs in dropsy, and in the neighborhood of inflammation and abscess. The muscles in these cases are not only bleached and discolored, but also macerated. A peculiar softening of the muscular coat to a gelatinous substance is sometimes produced by the action of the acids of the stomach and intestines: not unfrequently the process reaches beyond the stomach and involves the diaphragm.

§ 5. *Solutions of Continuity.*—In this class are included flesh-wounds of various kinds, and the lacerations and contusions of muscles produced by external violence; some of them are unattended by any injury to the integuments, whilst, in other cases, that and other structures also are more or less hurt. Muscles are subject to spontaneous rupture, giving way now and then during violent convulsions. The accident, however, is a rare one in any muscular structure but the heart. I have, on several occasions, met with ruptures of the recti abdominis muscles, which had been produced by the convulsions that occur in the course of ileo-typhus. Not unfrequently a few of the muscles of the limbs are found ruptured in the dead body, and the occurrence may be traced to excessive post-mortem rigidity. I have observed it most frequently in the biceps muscle of the arm. It may be distinguished as having taken place after death by the absence of any effusion of blood, and still more certainly by there being no trace of reaction.

The muscular coats of hollow organs do not usually escape laceration when the serous and mucous membranes, which invest them within and without, are violently torn; but sometimes the rupture is confined to the muscular coat. Not unfrequently it gives way in consequence of extreme distension, as for instance in the stomach, the intestine, and the bladder.

A wound of a muscle, though unattended by any loss of substance, can be repaired only in the same manner as one in which substance has been lost, for the retraction of the surfaces of the wound places the former in the same condition as the latter. Subcutaneous ruptures, those in which there is no corresponding injury to the superjacent integuments, and no exposure of the wound to the air, heal readily by the first intention: the surfaces become slightly inflamed, and a reddish, gelatinous exudation is effused, which fills up the space. It gradually changes into a reddish vascular cellular tissue, which at first unites with the surrounding inflamed cellular tissue, but afterwards separating again from it, is converted into a denser, white, leather-like (cellulo-fibrous) callus.

Flesh-wounds, which are complicated with injury to the integuments over them, and are consequently exposed, heal by suppuration and granulation. The granulations fill up the cavity of the wound, and changing into a dense white callus of the same kind, as in the former mode of repair, they unite the surfaces of the wound together. In such a case the inflammation extends beyond the wound for some distance along the muscle, and converts it into a cellulo-fibrous tissue, in which the muscular fibre disappears. The muscle is thus divided, and acquires two bellies; and not unfrequently a bursa is formed beneath the uniting callus, by which their movements are facilitated. In process of time this callus diminishes in size, partly by shrinking, and partly, perhaps, from being absorbed; and at length it may disappear almost entirely, a few traces of it only remaining.

In the stump, left after amputation, the extremities of the muscles become rounded off, and degenerate into a cellulo-fibrous tissue, and they unite in a common cicatrix with the integuments and thickened periosteum.

§ 6. *Diseases of Texture.*

1. *Hemorrhage.—Apoplexy.*—The various solutions of continuity, lacerations, and blows, which happen to muscles, give rise to effusions of blood within them: they are also liable to spontaneous hemorrhages into their parenchyma,—to extravasation of blood into their cellular and aponeurotic sheaths,—in fact to apoplexy. In cases of decomposition of the blood, trifling suffusions or ecchymoses occur in the muscles, as well as in the skin and subcutaneous cellular tissue; but in bad cases of scurvy and typhus, blood is extravasated from the capillaries rapidly and in large quantities, bruising and breaking the muscular fibres. Hemorrhages of this kind occur chiefly in the muscles of the lower extremities and abdomen, and are usually accompanied by effusions of blood into other tissues also.

2. *Inflammation.*—Inflammation of muscle (myositis) not only occurs as a consequence of the various injuries to which these organs are subject, whether incised, lacerated, punctured or gunshot wounds, or crushing, rupture, stretching, mechanical irritation, or concussion; but also arises spontaneously. It is sometimes a primary affection, and is brought on especially by the influence of cold; at other times it is a secondary consequence, either of metastasis, or of inflammation existing in adjoining organs. Its course may be acute or chronic. It is sometimes confined to a few scattered, and mostly small, spots in a muscle; at other times it invades the whole body of one, or of several muscles at once. As the cellular sheaths of the muscular fasciculi are almost always the seat of the process, especially if the inflammation be extensive, the distinguishing features of inflamed muscle will include those which characterize inflammation of cellular tissue. I must, therefore, necessarily refer to the latter, though attention will be chiefly directed to the change in the muscular tissue.

a. At first some redness and injection are perceived, and a little infiltration, of the interstitial cellular tissue; no change is discernible at this period in the muscular fibre itself.

b. But so soon as an actual exudation appears, the muscular fibre becomes discolored; it changes to a pale red, a reddish-yellow, or a fawn color.

c. If the inflammation continue, and the exudation be not absorbed, an important change soon takes place in the texture of the muscle; the fibres lose their transverse striæ, and the fibrils degenerate into a granular mass.

d. The exudation presents various, and essentially different characters, some of which are due to the particular cause of the affection, and some to the state of the constitution.

Some kinds of inflammation deposit, in the interstitial cellular tissue, and between the muscular fibres, a gelatinous, grayish or grayish-yellow, product, which gradually solidifies and becomes a whitish lardaceous callus; inflammations of this kind are mostly slow in their course, and are attended by mild symptoms: they are commonly named rheumatic.

The product of another kind of inflammation is reddish or yellowish, and fibrinous, and readily coagulates; it becomes converted into fibroid tissue.

Others, again, lead to a yellow exudation, which degenerates into pus; after a time the product exuded is a fluid, and either purulent or sanious. Inflammations of this character frequently destroy entire muscles.

Lastly, there are some inflammations, the product of which is essentially tubercular, and softens like tubercle.

Now and then, in cases of scurvy, and in inflammations of paralyzed muscles, a sanguineous product is found in the muscular tissue.

e. Under circumstances so various, the appearance of the inflamed muscle differs accordingly; its peculiar characters are most evident, when the inflammation is confined to isolated spots. The muscular fibres are then found in the midst of the exudation, discolored, disintegrated, and forming dull yellowish-red, or fawn-colored, and here and there interrupted, stripes. The exudation in which they lie may be a gelatinous, grayish, or grayish-yellow, substance; or firmer, and reddish or yellowish; or purulent and yellow; or tubercular and cheese-like; or red, filled with blood, and half coagulated.

f. An inflamed muscle is always somewhat shortened, and is paralyzed in that position; so that the limb in which myositis occurs, is nearly or quite immovable; and if the flexor muscles be affected, it is fixed in the bent position. The muscle is bound also in another way; for its cellular and fibrous investments having taken part in the inflammation, it becomes fixed in its sheath, and thereby also to other neighboring parts, which may have been inflamed with it.

Inflammation of muscle, if it do not subside, may terminate in induration, in suppuration, or in gangrene.

When an intense inflammation subsides, it leaves the muscle wasted; and if there have been extensive inflammations, the whole limb shrinks, and affords an example of secondary atrophy.

Induration is a mode in which inflammation frequently terminates; it is often found to have occurred at some isolated spots, especially in the heart. The exudation in the inflamed part coagulates and becomes converted into a whitish, lardaceous, firm callus, which assumes a fibroid structure, but is still traversed by a few pale and broken muscular fibres. The appearance of the callus varies according to the original quantity of exudation: at first it forms cords and streaks, which ramify amongst the fibres and fasciculi of the muscle, or more extensive, round, or nearly round, masses, which may be tolerably circumscribed, or may branch out rregularly in various directions. In the course of time, it may diminish n size considerably, partly from absorption, partly from its shrinking and becoming more dense, and from the disappearance of the muscular substance that still remains within it. As it thus diminishes in size, it draws in the surrounding tissue, and assumes the appearance of a deep cicatrix.

In a few cases this mode of termination has been found throughout the whole of a large muscle, and even in all the muscles of one or more of the limbs, particularly of the lower extremities: they were changed nto a white tendinous structure, and here and there into bone.

Osseous substance may be developed at a later period in the fibroid callus (*ossification*); and, in a few cases, it has been seen occupying entire muscles.

It happens, but very rarely, that induration of the muscular coat of an organ may be traced to a chronic inflammatory process. I have met with one such instance in the muscular coat of the bladder. It was changed into a pretty thick and firm lamina, of a uniform pale red color, inclining to yellow: the original bundles of muscular fibre had disappeared. It was closely united with the mucous membrane, but presented none of the peculiar change of texture characteristic of cancerous degeneration. The bladder was paralyzed and distended.

When inflammation of muscle terminates in *suppuration*, the matter may be formed at one or at several isolated spots; or if the process be extensive, rapid, and violent (tumultuous), it may involve the whole of the muscle at once. The abscess commences with the deposition of matter at several points; the intermediate muscular fibre becomes pale, or in some way discolored, and is dissolved; the points of matter coalesce, and the gradual union of small collections of this kind at length destroys the whole muscle. When the suppuration runs a chronic course, the cellular sheath and outer layer of the muscle very commonly change into a thick lardaceo-fibrous membrane: in fact the muscle is converted into a thick-walled bag of matter. Certain muscles are particularly liable to be destroyed in this manner, such as the psoas, and the iliacus; and the disease is well known in the instance of the former muscle by the name of psoas abscess. The sac of the psoas abscess is spindle-shaped or cylindrical, and it is lined sometimes by a soft, diffluent, shreddy tissue, which is soaked in pus or sanies, sometimes by a smooth, grayish-red, or grayish lamina, resembling jelly; it may be full and tense, or collapsed, according to circumstances. At the insertions of the muscle into the bodies of the vertebræ, the sac not unfrequently spreads out into sinuses or pouches, while near the tendon it contracts like a funnel. Rounded bands of softened, or of lardaceo-fibrous and degenerated muscular substance, pass like bridges across the cavity; they take various directions, but are, for the most part, longitudinal. The crural vessels and nerves pass over the sac externally, and are stretched by, and adherent to it. The crural vein is not unfrequently found inflamed, and stopped by a plug of blood. In the further progress of the abscess, the centre of the tendon suppurates, and the sac, thus enlarged, appears below Poupart's ligament, and extends at last quite to the insertion of the tendon.

If the patient does not die from exhaustion at an earlier period, ulceration takes place in the wall of the abscess and the adjoining parts; the matter gravitates in different directions, and is occasionally discharged externally. Caries of the ilium may ensue; the matter may pass under the fascia and among the muscles of the thigh, or towards the great sacro-sciatic foramen; and the abscess may open at the inner side of the thigh, in the abdomen above the groin, in the nates, &c.

In favorable cases the abscess heals up: the matter having been discharged or absorbed, the sac contracts and shrinks to a fibroid (ligamentous) cord, which sometimes retains in its interior chalky remains of inspissated purulent matter.

There is a very frequent combination with this abscess of muscle, and one of considerable importance, the relation of which to the suppuration in the muscle still requires explanation; I allude to caries of the bone into which the upper end of the muscle is inserted. Caries of the ilium, in cases of psoas abscess, is unquestionably consecutive; not so, however, caries of the lumbar vertebræ. This combination of disease in muscle and in bone, is certainly most frequently observed in the instance of the psoas muscle and the lumbar vertebræ; but it is sometimes found existing between the spine, and the tendinous crura of the diaphragm or the long muscles of the neck. I have met with it in other parts also, where muscles are inserted into spongy bones; for instance, at the insertion of the sartorius and gracilis, I have found the bone inflamed, and suppuration commencing in the interior of the tendon. The process is very rarely seen at its commencement; but from an analysis of the combination, as it has occurred to different observers, it seems that in some cases the inflammation and suppuration of the muscle is the primary disease, and the caries consecutive; whilst in others, the bone is first affected, and the abscess is formed chiefly by the subsequent collection and gravitation of the purulent matter.

From what I have had an opportunity of observing, it appears to me extremely probable, that in many cases the bone is the first diseased, whilst in many others the affection commences simultaneously in both structures; in the latter case it usually happens, that the suppuration in the tendon of the muscle spreads, especially at first, with most rapidity.

a. The caries, thus combined with abscess, is almost always of tuberculous nature; as, therefore, tubercle is a very common disease in bone, but is very rare in muscle, it is so far likely that the bone would be first diseased and the muscle afterwards.

b. But, on the other hand, I have met with cases which support the view, that the affection begins simultaneously in the bone and muscle,—cases in which at the insertion of the sartorius, the gracilis, or the long muscles of the neck, the bone was inflamed, and suppuration was commencing in it, whilst the tendon of the muscle formed a funnel-shaped abscess around the diseased spot of bone. Moreover it may be presumed, that this is the way in which the inflammation would extend to the muscle, when the bone is primarily affected.

When the disease has commenced in the manner last described, it extends into, and produces caries in the bone; while on the other side, advancing from the point of the funnel-shaped cavity, it excavates the belly of the muscle: as it spreads it leads to the deposition of a tuberculous exudation, and reduces the muscle to a mere abscess, which communicates through the narrow sinus in the tendon with the ulcerating cavities within the bone.

The contents of the abscess are tubercular, purulent matter, and fragments of necrosed bone, infiltrated with tubercle.

3. *Metastasis in muscles.*—Deposits of purulent and sanious matter are sometimes found scattered extensively throughout the muscular system; they occur for the most part when pus or sanies has been taken into the mass of the blood.

4. *Gangrene of muscles.*—Sometimes inflammation terminates in gangrene; the muscular substance becomes much discolored, and changes into a shreddy, almost diffluent mass, soaked through with sanies, and of a greenish-brown color.

The state of the muscular substance is peculiar beneath an eschar, and in cases of mummification. It is converted either into a dirty reddish, soft, humid, spongy, pap-like pulp, or into a more dry, tinder-like and fragile mass, that shrinks and crumbles together.

5. *Morbid growths.*—The muscular system is rarely the seat of morbid growths, except when it is involved in those which have originated in other tissues.

a. Teleangiectasis.—It occurs under the form of more or less extensive convolutions of dilated vessels, by which the muscular substance becomes displaced and atrophied, though at the same time the belly of the muscle retains its natural outline.

b. Formation of fat.—What is called conversion of muscle into fat, or steatosis musculorum, is a change into a substance resembling adipocere or spermaceti, or into actual fat. The muscles most exposed to it are the voluntary muscles, especially those of the lower limbs: slighter degrees of the same change affect also the muscular substance of the heart, and the fleshy coat of the gall-bladder. There are two forms in which it occurs.

a. In the first, adipose tissue or fat-cysts are formed between the ultimate muscular fibres; in the voluntary muscles the transverse striæ[1] disappear, and their red color is exchanged for a paler hue, a pale yellowish-red, a dirty yellow, or a dun color: after a time the fibres themselves become disintegrated and disappear, and the muscular tissue gives place altogether to a mass of fat. Small globules of free fat are found as well as fat-cysts; and sometimes there is also a yellow granular pigment.

Many grades of this degeneration have been observed, which run imperceptibly into one another. They are distinguished by the external condition of the diseased muscle, which varies in respect to the amount of discoloration, the degree in which its texture is lost, and the preservation or alteration of its external form.

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[1] It is not yet made out whether the disappearance of the transverse striæ is a primary or a secondary part of the change; but it is probably the former; for they disappear in paralyzed muscles, under the same conditions as those under which steatosis takes place, though steatosis be not present.

The earliest change is one of color. The muscle has a pale reddish appearance; and is found, on close examination, to be not uniformly discolored, but stained irregularly of a yellowish or fawn color, as well as marked with longitudinal pale reddish streaks, which follow the course of the fibres.

As the stains of fat increase in size and coalesce, the muscle acquires an almost uniform fawn color: but its fibrous arrangement still remains distinct.

With the advance of the disease it becomes altogether of the color of fat; sometimes being yellow, sometimes remarkably white, and resembling accordingly either tallow or spermaceti. No trace of its fibrous structure remains except some of its tendon, or the cellular sheaths of its fasciculi.

Up to this stage of the disease the outline of the muscle has been preserved; but in the last stage the mass of fat, into which the muscle is changed, mixes with the adipose structures around it, with the subcutaneous adipose tissue, or with other masses of fat which have been developed by the same process in adjoining structures. We may then find in a limb nothing of its muscles, but remnants of tendons, and aponeuroses with their prolongations inwards.

A muscle thus diseased is usually very thin; and an extremity in which all, or a great part of, the muscles have undergone such a change, is slender as if emaciated, and cylindrical from losing the contour of its muscles: it has also a uniform feeling of toughness.

These are the stages of the disease in question, from that at which it is first distinguishable by the anatomist, to that in which the muscle has completely disappeared, at least so far as opportunities of observing it have occurred to myself and others. In its first and slightest degree it is closely allied to that state of emaciation and pallor in which the animal muscles are found when there is a great and general deposit of fat in the body, such as occurs chiefly in the female sex, and is associated with changes in other organs, which will be mentioned afterwards. It is allied, also, to the state of atrophy of the organic muscular coat of the bowel, and even of the bladder, &c., which is found when large quantities of fat are accumulated in the folds of the peritoneum and cavity of the pelvis.

The process consists, therefore, in the production of a quantity of fat, which compresses the muscular fibres; and the name of fatty metamorphosis of muscle can be applied to it only in so far as the place of the muscle is actually occupied by fat.

Whether the disease occur in one muscle or in many, it usually commences at every part of them at once. Sometimes, however, it appears first at several isolated spots on the surface or in the interior; or it terminates at particular points, as is especially the case in the heart.

It is met with in the substance of the heart, and in the fleshy coat of the gall-bladder, but is most common in the muscles of the lower extremities.

Among the causes which lead to it, we are able to enumerate advanced age, a sedentary luxurious kind of life, the misuse of alcohol, and complete inaction of the muscles,—such inaction as is produced by anchylosis, or by spasmodic contraction of some other muscle.

The fatty degeneration which is brought on by a sedentary and luxuous mode of living, and by spirit-drinking, is, for the most part, accomanied by a great development of fat throughout the system; the liver sually contains a quantity of tallowy substance, the heart is loaded ith fat, and its muscular tissue is more or less metamorphosed into the me substance: moreover, in old people more particularly, the medulla f the bones connected with the altered muscles is in excess, and the ones are in a state of osteoporosis, or excentric atrophy, and are easily roken.

The fat by which the muscular tissue is supplanted varies in its chacter. In some instances it resembles ordinary healthy fat; sometimes, pecially in persons advanced in years, it is of a dark yellow color, ose and diffluent; and sometimes, in its consistence and whiteness, it remarkably like mutton suet. It assumes this last character, particurly when the change results from anchylosis.

β. There is another form of degeneration which has not been hitherto bserved, and to which the muscular structure of the heart is liable, parcularly when hypertrophied: it is met with, also, but not so frequently, the muscular coats of other organs when they are hypertrophied, but very seldom seen in the muscles of animal life. It is characterized by e development of minute particles of free fat between the primitive uscular fibres. At the same time the striated sheath of the fibre disppears, and the muscle changes to a dirty yellow or fawn color, and ecomes friable. I once met with the disease in the muscles of the calf, which it had given rise to considerable pain: this fact coincides with e experience of other observers.

c. Cysts.—With the exception of cysts which enclose entozoa, these rowths are very rare in the muscular system. Even the large sized ephalocyst sacs are very seldom found. And this recalls the fact, at cancerous growths also are extremely uncommon in muscles, although comparison of this sort leads to no result, inasmuch as muscles are rely the seat of morbid growths of any kind.

d. Fibroid tissue is found in muscles which remain indurated with llus after inflammation, and in those which are spasmodically conacted.

e. Bony growths (1,) not unfrequently exist in muscles in the form of lcareous concretions, which have been developed in the fibroid tissue st mentioned. In some few cases, whole muscles have been found thus sified. Muscles sometimes contain also the cretaceous remains of pus, tubercle, and of the shrunken sacs of the cysticercus and acephaloyst (echinococcus).

(2.) True bone is less frequently met with in muscles. When it occurs, assumes a rounded form, or is flattened and elongated, but still rounded. ometimes it is spongy, and sometimes of more compact structure. he drilling bone (Exercirknochen), as it is called, in the left deltoid uscle, is of this nature, and numerous other growths of the same kind e to be met with. In the museum at Vienna, a very large, egg-shaped, iece of bone is preserved, which was taken out of the biceps of a woman's ft arm.

f. Tubercle.—Primary tubercle scarcely ever occurs in the muscular system, least of all in the form of gray granulations. Even those inflammations of muscles which have been already mentioned as leading to tubercular deposition, and as connected with similar affections of bone, are, generally, secondary inflammations; they almost always occur in combination with tuberculosis of parenchymatous organs, and especially with already established tuberculosis of the lungs. Under similar circumstances, and according to my observations when phthisis already exists, inflammations now and then arise independently of diseased bone, in the interior of the bodies of different muscles: they furnish an exudation of tubercular nature, and as this breaks down, they give rise to the formation of a tubercular vomica in the muscle.

But muscles sometimes become the seat of tubercle and tubercular softening, in consequence of their vicinity to other affected organs; and this is especially the case with organic membranous muscles, particularly with the fleshy coat of the intestinal canal. The fact is seen in the muscles surrounding the thorax when there has been tubercular softening of the lung and pleura, in the muscular coat of the bowel beneath a tubercular ulcer of its mucous membrane, &c.

g. Cancer.—In whatever form this disease presents itself, it is scarcely ever the primary cancerous affection in any muscle of animal life, except the tongue. One or more cancerous growths are almost always found elsewhere, and that in the muscular system is the secondary affection. Of those organs even which are entirely composed of organic muscular fibre, nearly the only one which is ever primarily attacked by cancer is the uterus; and it is the cervix and vaginal portion of the uterus,—the muscular development of which is in the unimpregnated state very subordinate to that of its body,—that is especially subject to the disease. But muscular tissue, is more frequently involved in the degeneration of adjoining organs affected with cancer, and to this secondary affection both animal and organic fibre is subject; the pectoral muscle, for instance, in cancer of the breast, and the muscular coat of the alimentary canal, in cases of cancer of the stomach or bowel.

The forms of cancer which are most frequently observed thus attacking the muscular system, are the fibrous and medullary.

A firmly fixed knotted tumor, inwoven with the muscular tissue, and sending out branches in all directions, is sometimes the distinguishing character of the fibrous kind of cancer; but more frequently, when the muscle is diseased in consequence of its proximity to some other affected organ, it produces a characteristic degeneration of the muscular tissue. This degeneration is mostly seen in cases of secondary disease of the pectoral muscle, or middle coat of the intestines, and it leads to the conversion of the muscular tissue into a white, fibrillated, reticular structure, in the interspaces of which a pale reddish, or yellowish-red substance resembling firm jelly is lodged. The muscular coats of organs, under such circumstances, are palpably increased in size. Degeneration of the same kind is observed not only in fibrous, but also in medullary cancer.

The medullary form is not unfrequently associated in muscles with a very extensive growth of cancer. It sometimes presents its genuine white character, sometimes that of melanosis; and it grows in rounded, circumscribed, encephaloid knots in one or in several muscles. When the muscle is diseased in consequence of the degeneration of adjoining organs, encephaloid matter seems to be infiltrated throughout it amongst the muscular fibres, some of which are blanched and others degenerated in the same manner as in fibrous cancer.

Like other structures, muscle sometimes resists for a long period the advance of large cancerous growths; it becomes thin and atrophied from the pressure and stretching, but undergoes no actual change of texture.

h. Entozoa.—The entozoa which occur in the muscles of the human subject, are—

α. The echinococcus, which inhabits the acephalocyst. It is seldom met with. The sac is situated between the fibres, and forces them more or less asunder.

β. The cysticercus (Blasenschwanzwurm) is somewhat frequent. It is very often found in several or in most of the muscles, as well as in the heart of the same individual, and not very rarely in the brain too. The number existing in a single muscle, and in one individual, is sometimes quite extraordinary. The cysticercus seems to share with the trichina spiralis the remarkable character of being confined to voluntary muscles (muscles with transverse striæ); the distinction is not perhaps so strictly marked as in the instance of the trichina, but it has been observed in several cases.

When the cysticercus dies, its tail-vesicle shrinks, and the contents become inspissated, and at length cretaceous. Chalky concretions enclosed in a thick cyst are often found in muscles, which are the remains of cysticerci.

γ. The trichina spiralis is an entozoon which is strictly confined to the voluntary muscles. Upon its death it leaves an encysted chalky concretion behind.

i. Foreign bodies.—All kinds of extraneous bodies are introduced into muscles by natural or unnatural means, such as needles, bullets, fishbones, &c.

PART XII.

ANOMALIES AND DISEASES OF THE NERVOUS SYSTEM.

PART XII.

ANOMALIES AND DISEASES OF THE NERVOUS SYSTEM.

The abnormal conditions of the nervous system may be subdivided into those of the brain, those of the spinal cord, and those of the nerves. To the description of the two former I shall prefix an account of the disorders of their investing membranes.

CHAPTER I.

THE BRAIN.

SECT. I.—ANOMALIES AND DISEASES OF THE MEMBRANES OF THE BRAIN.

The membranes of the brain become involved both in faulty development, and in structural diseases of the cranium on the one side, and of the brain on the other. They are liable also to many idiopathic diseases.

DURA MATER.

§ 1. *Deficient and excessive development.*

1. This membrane is sometimes entirely wanting in consequence of the absence of the brain; and portions of it are deficient, when the development of the brain is in any way arrested; thus the tentorium or the falx is sometimes wholly or partially wanting.

2. When the brain is double, there are two more or less complete sacs of dura mater.

§ 2. *Anomalies in Size, Form, and Position.*—These include the unnaturally small or large capacity of the sac of the dura mater, which results from corresponding anomalies in the formation of the brain, whether want of symmetry between its two halves, or displacement of them from their natural situation; or which results from an unnatural direction of the falx, from partial dilatation and (hernial) protrusion through the cranium, &c. And at any period of life, the dura mater may be distended and more or less attenuated, and its internal processes displaced in various directions by hypertrophy of the brain, hydrocephalus, morbid growths in the brain, &c.

The thickness of the dura mater is often manifestly increased, not only in consequence of inflammation, but also in persons who are advanced in life. Sometimes also it becomes thin, either in its whole extent, as when the brain is hypertrophied; or at certain parts where it is subjected to pressure; thus it is found thin and cribriform where it has been exposed to the pressure of the Pacchionian bodies.

§ 3. *Solutions of Continuity.*—Under this head we class together anomalies in the adhesion of the dura mater to the cranium, and solutions of the continuity of the membrane itself.

Besides the thickening which the dura mater undergoes in advanced life, it acquires also a closer adhesion to the cranium; for as new bony matter is at different periods deposited on the inner surface, especially of the vault of the cranium, and the number of vessels with their investments passing between the membrane and bone becomes increased, the union between them is rendered more intimate. The adhesion generally commences, and continues strongest, along the sutures. In very old age the adhesion is often remarkably less firm than it is in middle life; the cranial bones are then atrophied.

The adhesion between the dura mater and cranium is frequently rendered much looser, or even entirely broken for a more or less considerable space, by concussion of the skull; the separation may take place at the part where the blow was struck, or on the opposite side of the head. Its extent is often increased by subsequent extravasation between the membrane and the bone. Purulent and sanious effusions gradually force the dura mater and the bone apart.

The dura mater and cranium are frequently found separated from one another in cases of cephalhæmatoma, by an extravasation of blood, which takes place spontaneously from the bone.

Various kinds of solution of continuity befall the dura mater from incised, punctured, and gunshot wounds, and from injuries which break and shatter the skull. The membrane may split also, from being much distended by pressure from within in cases of hydrocephalus; and these ruptures of the dura mater are not limited to cases of hemicephalus; in some extremely rare instances, rupture occurs in hydrocephalus during extra-uterine life. Such a case is preserved in the Vienna Museum; it is a rupture of the dura mater, near the right parietal protuberance, more than an inch in length; it occurred in a hydrocephalic boy, and was followed by the extravasation of blood and of serum from the ventricle, between the dura mater and the cranium, and thence under the pericranium.

The Pacchionian bodies very often exert such pressure on the dura mater, as to give rise very gradually to solution of its continuity and concomitant atrophy. They force the fibres of that membrane asunder, and having pressed through it, lie in immediate contact with the skull.

Somewhat rarely it happens, that the dura mater is forcibly separated into layers by an extravasation of blood within it after concussion of the skull: such a separation is still more rare as a consequence of suppuration. Those extravasations which have been supposed to be collections of blood between the serous and fibrous strata of the membrane, with the exception of a few cases in which a small effusion has raised its innermost layer, must have been extravasations into the sac of the arachnoid, which, after acquiring an adhesion to the dura mater, have become ncysted: they will be considered among the diseases of the arachnoid.

§ 4. *Diseases of Texture.*—A distinction will be drawn in the followng remarks, wherever it is possible, between the actual dura mater, and ts innermost shining stratum. For, though the latter cannot be demonstrated as a separate serous layer, we are compelled to adopt the distinction by the substantial difference which is exhibited, at least at first, by morbid processes in the two layers. Inflammation, for instance, attacks one of the layers independently of the other, and presents differences accordingly in its course, in its proneness to extend along the surface, and in the products it furnishes, which manifest the analogy between that layer and serous membranes in general. I shall not at present enter into these, or into several similar subjects, as it would be nterfering with diseases that are evidently connected with the arachnoid membrane.

1. *Inflammation.*—With the exception of those which are brought on by injury, primary inflammations of the dura mater to any extent, such, for instance, as would lead to the formation of matter, are of rare occurrence.

Inflammations of slight degree, on the other hand, and usually combined with moderate inflammation of the cranial bones, are frequent. These processes are characterized by vascularity and rosy reddening of the dura mater, and by softening of its texture; and they give rise to nterstitial infiltration of the membrane, as well as to exudations upon that surface of it which adjoins the bone: such exudations become organized nto loose cellular, or thick fibrous tissue, or at length, especially if there be any inflammation of the bone besides, into bone; and they produce an unnatural adhesion to the vitreous table of the skull. They are generally widely spread, especially along the sutures; but sometimes they are confined within a smaller compass, so that they form at one time extensive tracts, at another circumscribed islands. Exudations which, ossify, very commonly present themselves spread out as a layer, that, ike the puerperal osteophyte, is at first spongy, but gradually becomes compact: sometimes they form a mass of bone which looks as if it had lowed or dropped upon the membrane, and then coagulated; while, not unfrequently, they are circumscribed osseous plates or nodules, which, though, in course of time, intimately united to the bone, yet originally adhered firmly to the dura mater.

When the inflammation is more intense, and runs a chronic course, the dura mater acquires an increase in thickness, sometimes to the extent of three lines, and even more; it becomes indurated and callous, and usually adheres more closely than natural to the bone. I met with an nstance of this effect of inflammation in the dura mater lining the right occipital fossa, in which inflammation came on in the adjoining lateral sinus, and led to its obliteration.

When inflammation is brought on by injury, or passes to the dura mater from neighboring tissues, it frequently terminates in the production of matter, and in suppurative degeneration of the membrane.

These latter cases, therefore, are of great importance, for they are brought on by inflammation and suppuration of the bone, or of neighboring ligamentous structures. They are especially apt to occur in particular localities; thus the dura mater inflames, suppurates, and sloughs from caries of the internal ear, and the labyrinth of the ethmoid bone, from caries of the upper cervical vertebræ, and suppuration of their ligaments. In the dura mater these processes continue circumscribed, but when they reach the inner membranes, they usually spread rapidly into general meningitis.

The characteristics of inflammation of the dura mater are those which are common to all inflamed fibrous tissues: I refer on this point to what has been said at page 94.

2. *Adventitious growths.*

a. Cysts.—Cysts properly belonging to the dura mater are extremely rare; though some examples have been met with in its substance of fat-cysts containing hair. I have in some cases seen tumors attached to the inner shining surface of the dura mater, which resembled lipoma; they were enclosed in a cellular sac, and more or less distinctly lobulated.

b. Fibroid tissue.—Besides the fibroid thickening of the dura mater which is found after inflammation, tumors of fibrous structure occur in that membrane. They are, however, very rare; in a great number of tumors I remember only some few which exhibited a genuine, developed, and undoubted fibrous structure. Very many growths in the dura mater have indeed an intermixture of fibres with their structure, differing in various cases in arrangement and degree, which gives them the appearance of a fibrous tumor. There is no doubt that the tumors on the petrous bone, particularly examined by Cruveilhier, were structures of this class supposed to be purely fibrous.

c. Productions of bone.—What are called ossifications of the dura mater, are known to be very common. By these are generally meant the bony growths situated on the inner surface of the dura mater, especially of the falx. I do not, however, believe that these belong to the dura mater itself. From their analogy with similar formations on the cerebral, and especially on the spinal, arachnoid, as well as from special examination of their relations, I infer that they are connected with the arachnoid layer of the dura mater. They will be noticed more completely among the abnormal conditions of that membrane.

There are, however, some bony formations which undoubtedly belong to the dura mater. The osseous plates before alluded to, which become united to the cranial bones, and which are true bone, and the bony concretions sometimes developed in the dura mater, when it has become thickened and callous from inflammation, are of this kind.

d. Tubercle.—Primary tuberculosis is as rare in the dura mater as it is in the fibrous system generally. The tubercles, which are often found adhering to the inner surface of the dura mater, are such as were originally developed in the peripheral cerebral substance, and subsequently came into connection with the dura mater by the formation of adhesions between them and the inner membranes. They do not belong, therefore, to the present section. On the other hand, the dura mater is not unfrequently the seat of tubercular deposit and tubercular suppuration, when the cranial bones are carious from tubercular disease.

e. Carcinoma.—Even excluding from consideration the various growths of cancerous nature, and those more or less allied to cancer, which present themselves on the internal shining surface of this membrane, we yet find cancer of the actual dura mater to be of rather frequent occurrence. It sometimes exists primarily, that is originates, in the dura mater; but much more frequently the membrane becomes diseased secondarily by its contiguity with the cranium,—cancer of the bone implanting itself in the dura mater after having perforated the vitreous table. Even cancers of the brain sometimes implicate the dura mater.

Primary cancer appears—

α. In the form of cancerous infiltration of the tissue of the dura mater, in tracts which are mostly considerable in extent. The membrane is thickened to a corresponding extent, and the surface next the bone is often covered with a layer of cancerous matter, which makes its way into, and destroys the cranial bones: at length the disease appears also on the inner surface of the dura mater, destroys its arachnoidal layer, and spreads out in the sac of the arachnoid in the form of one uneven rugged stratum, or of separate nodulated protuberances. It is always combined with cancer in other organs, and assumes the form of white encephaloid, or of cancer marked with black pigment.

β. In the form of rounded tumors, which commence in the fibrous tissue of the dura mater, and protrude, as they grow, either outward or inward, or in both directions. It is sometimes found alone; very commonly it is of the medullary kind; it is often characterized by fibrous arrangement, and often, too, by considerable vascularity; or it resembles in its structure the parenchyma of some glands.

This last form exactly corresponds to the morbid growth which has been, and indeed is still, known by the name of fungus of the dura mater. The question, as to whether the dura mater or the calvarium be its primary seat, is no longer important, as it is settled in what has just been said: and the discussion which has been carried on upon the subject has now only an historical value.

If the fungus grow outward, it makes its way, by the pressure and absorption which it occasions, through the skull, and appears under the integuments: after a time it perforates them also. The loss of substance is more extensive at the inner than at the outer table; and as the fungus, after having perforated the skull, swells out and grows without restraint, it is girt by the sharp bevelled margin of the bone, and a constriction, or kind of neck, is produced. This condition of parts has been looked upon as very characteristic of these cases, it is, however, far from being universal; it is not found, for instance, when the bone degenerates together with the fungous growth, when it is by the malignant degeneration of the bony tissue that the fungus makes its way to the surface; still less is it found in cases in which the fungus of the dura mater was originally a fungus of the cranium, or in the stage of softening of the fungus, in which the aperture becomes enlarged by corrosion of the bone.

The usual situation of cancer of the dura mater is the vault of the skull; it seldom happens at the base. If it spread as a fungus inward, the bone beneath it commonly becomes thickened and dense, or the fungus springs from the dura mater just over a plate of new bone.

THE ARACHNOID.

The arachnoid is a shut sac, the visceral or cerebral layer of which is, for the most part, blended with the pia mater in the same manner as serous membranes are with the tissue which lies beneath them: but in some parts, which vary in extent, it departs from this relation to the pia mater, and is thereby distinguished from serous membranes; that is to say, by being unconnected with any subjacent tissue, and by having a double smooth surface. And corresponding to this peculiar anatomical arrangement, there are various features in diseases of the inner membranes (arachnoid and pia mater), when regarded as one organic whole, by which they are distinguished from diseases of other serous membranes. It is desirable that the diseases of the arachnoid should be considered apart from those of the pia mater, because the former, especially in its parietal layer, is subject to many affections which are peculiar to itself, and because the diseases of its visceral layer also are in many respects independent of the pia mater: moreover, there appears to be less confusion in studying the relations of two things in separate and adjoining sections, than in overwhelming one section with differences and peculiarities, and speaking sometimes particularly of one, sometimes of the other, and sometimes of both.

§ 1. *Anomalies in size.*—To this head belong congenital enlargements of the arachnoid; they are produced by what is called external or meningeal hydrocephalus, and are either partial (local) or general. The former are cases in which sacs of a dropsical arachnoid protrude through an aperture in the skull. I shall have to treat of these cases among the Anomalies of the contents of the arachnoid sac.

§ 2. *Diseases of Texture.*

1. *Hyperœmia.*—We very rarely have an opportunity of seeing the vessels of the arachnoid in a state of congestion, or, indeed, of detecting any injection of them that is perceptible with the naked eye. But changes, nevertheless, are frequently discovered in the membrane, which can be attributed only to congestion, or to slight and passing attacks of inflammation, and which are always accompanied with changes in the pia mater of a similar nature, and arising from the same source.

These changes increase in degree with each recurrence of the congestion, but they are found to be most marked when it has become habitual. The cerebral arachnoid is the most frequently affected, especially that portion of it which covers the convexity of the cerebral hemispheres. The changes consist in opacity, thickening, and hypertrophy. Sometimes large extents of surface are pretty uniformly affected, at other times separate spots are more prominently, or are alone altered. The arachnoid tissue is opaque, dull like whey or milk, tumid and white, and it has the appearance and density of tendon. One form which the affection assumes is remarkable for its frequency; it is well known by the name of the Pacchionian bodies. They are merely a granulated form of fibroid thickening of a serous membrane. The granulations are found oth singly and in groups, and when they exist in any number, they are enerally situated upon an arachnoid membrane that is cloudy and thickned. By their pressure they force the fibres of the dura mater asunder, nd become imbedded in it; then perforating that membrane, they occupy mall pits and fossæ of their own in the cranial bones, and in this manner ive rise to an unnatural adhesion between the cerebral arachnoid and he dura mater. They are usually found at the margin of the hemipheres adjoining the falx, and in that situation they often perforate the vall of the superior longitudinal sinus, and project within its cavity.

Opacity and thickening of the arachnoid are very common post-morem appearances: after middle life, a moderate degree of them is almost onstantly found, and their absence is the exception; for at that period very one must have been exposed to repeated congestions of the brain nd its (inner) membranes. This is especially true of the Pacchionian odies, which, as is well known, are scarcely ever absent, even in earlier ife. In persons who have suffered from frequent, intense, and protracted ongestions, they are more considerable; but the most marked examples ucceed the congestions produced by frequent intoxication, and repeated ttacks of delirium tremens.

Moreover, the arachnoid membrane is found augmented in actual bulk, nd containing within its cavity an increased quantity of serous effusion, n cases of atrophy of the brain, and, indeed, in combination with various ther appearances, which are all occasioned by the tendency to a vacuum vithin the cranium, and will all be detailed in the sequel.

2. *Hemorrhage.*—Spontaneous extravasations of blood into the sac of he arachnoid are by no means uncommon. They mostly happen on the onvex surface of the hemispheres; at least, in the more extensive effuions, it is always in this situation that the largest quantity of blood is ccumulated; small effusions frequently take place at the base of the kull, but larger ones occur there very rarely. They are seldom met vith while in the recent state; and from their having usually existed for long period before they are examined, the effused blood is found to ave already undergone considerable changes. I will begin by depicting well-marked example, and afterwards analyze its appearances.

Lying beneath the dura mater that covers one of the hemispheres, is ound a sac or cyst, which resembles in form a flattened cylinder, somewhat curved from before backward in correspondence with the arch of he cranial vault, or resembling in shape what results from the forcible eparation of two layers of a tissue by an effusion which commenced at ome single point, and then spread out between them. The figure first lescribed involves an excess in the measurement from before backward ver the transverse and the vertical diameters. The sac adheres by its uter surface to the dura mater, but its inner wall is free, or nearly free, rom any connection with the cerebral arachnoid, and is consequently nore or less smooth, and moist. Its adhesion with the dura mater, too, s but loose; it partly sticks on, and partly is connected with the membrane by a few small vessels. Both walls of the sac are usually of a rown, rusty color, and tenacious; they may often be separated into everal layers, which vary in thickness, but the inner of which are the nore thin: at the margin of the sac they coalesce and form one lamina, which soon becomes reduced to a thin, brown, rusty-colored membrane, and spreading out further on the cranial vault, reaches to the base, and at length terminates in a thin, rusty-colored, gauze-like film. Loose shreds of plastic lymph hang on the inner surface of the walls, and (which is remarkable) principally on the wall which adheres to the dura mater; within these the sac contains a more or less thick fluid, of a dark and various color, like chocolate, or plum-sauce, rust, or yeast: in course of time the lymph is gradually removed, the inner surface of the sac becomes smooth and polished, and the contents are changed into a colorless, thin, clear, serous fluid. The corresponding hemisphere becomes plane or slightly hollowed, its convolutions flattened, and its ventricle narrowed, while the serum of the ventricle is forced over into that of the opposite side. The pressure of the cyst sometimes diminishes the thickness of the parietal bone.

On closer examination, the outer layers of the wall of the sac are found to be vascular. At the margin of the sac, just where its two walls unite into one stratum, numerous small trunks may be seen, sending branches and twigs both upon and into the walls, especially that wall which adjoins the brain. Here and there also other vessels pass into the convex surface of the sac from the dura mater. The walls are composed of plastic lymph, and contain various quantities of modified red particles of the blood; in their outermost layers fibrils like those of cellular tissue are being developed. The coagulation of this lymph at the periphery of the effusion, is the occasion of its becoming encysted.

These sacs are usually borne for a long time, and they do not present any proof of having diminished from their orginal size; for the impermeability of the wall of the sac materially interferes with the absorption of its fluid contents. Occasionally, however, cases do occur, in which a sac that originally was of large size, has undoubtedly become smaller, some of the fluid part of the contents having been at length absorbed, and the cavity diminished by the approximation of the two walls to each other, and by their cohesion at the margin of the sac. In some cases it has even completely closed in this manner, and is wasted.

The walls of the sac, especially that one which adheres to the dura mater, sometimes become the seat of ossification, that is, of bony concretions in the form of plates.

In a considerable number of observations, I have seen but one case in which the sac was of a cylindrical form, and filled, like a sausage, with a dark, reddish-brown plug of coagulated blood.

Some rare instances occur of large circumscribed extravasations into the arachnoid sac, in which the fibrin does not disengage itself, and coagulate at the surface, and thus enclose the extravasation; such cases present to view a rounded and irregular mass of coagulum.

Small effusions, which spread out and form a thin stratum, leave behind them a correspondingly thin, single lamina, of a rusty brown, or yeast-yellow color, which lines the dura mater. It is very often a mere film, and can be detected only by the closest examination.

According to my observations, these extravasations occur pretty often in adults, and especially during and after the best years of manhood: Barthez, Rilliez, and Legendre, have not unfrequently noticed them in young persons and children. It has been my uniform experience, that the large encysted extravasations over the hemispheres, induce a marked degree of weakness of intellect. They have much general interest, as rare examples of hemorrhage into a serous cavity. The source of the bleeding cannot usually be discovered. In persons who are advanced in life, it may, perhaps, arise from the congestions which are occasioned within the skull, by atrophy of the brain.

It should be remarked, that old effusions may be mistaken for hemorrhagic exudations; but such an error may be obviated by observing the following particulars:

α. At whatever period the extravasation may be examined, no change of structure, such as accompanies inflammation, will be found in the arachnoid, even in its parietal layer, to which the extravasation is attached.

β. In the recent state, it is clear that the effusion is pure blood.

γ. Further distinctions may be noticed, in the peculiar change of structure which has been described as taking place in the extravasated fibrin, and in the want of any intimate organic connection between the sac and the dura mater.

Lastly, it is these encysted extravasations which have hitherto, for a long time, been erroneously looked upon as extravasations between the two layers of the dura mater.

8. *Inflammation* (*Arachnitis*, *Arachnoditis*).—The condition of the pia mater subjacent to an inflamed arachnoid membrane, produces sundry peculiarities in inflammation of the cerebral layer of the arachnoid.

In the first place, inflammations of the parietal layer of the arachnoid are, on the whole, of frequent occurrence; their pathological import, however, is mostly subordinate, for they are a secondary appearance, or, as it were, a complementary localization of the general disease; they occur in the course of processes attended with extensive exudations, particularly of those which take place on serous membranes; they are met with also, in cases of what is called a phlogistic state of the blood, and in pyæmia, in the course of acute exanthemata, of Bright's disease, of acute biliary dyscrasia, &c.; and they are commonly slight in degree. The inner surface of the dura mater appears streaked with delicate vessels, and is of a clear, rosy red tint; it is lined with an exudation, that may be delicate, grayish, and soft, like a layer of mucus, or more consistent, and membranous, or yellow, loose, and puriform.

In some rarer cases, the inflammation of the parietal layer has all the appearance of being primary, and, judging from the amount of its products severe also. The inner surface of the dura mater is found lined with a false membrane, from half a line to several lines in thickness; it is of cellular structure, and is mostly remarkable for its vascularity, and for a corresponding degree of redness. It is attached to the dura mater by means of its vessels. Sometimes it contains yellow cheese-like masses of disorganized fibrin, which vary in size, and in shape are rounded or irregular, and branching and coalescing. Still more rarely, the exudation becomes a dense, fibroid membrane, in which plates of bony concretion are developed, as so often occurs in false membranes on the pleura.

These processes usually take place, and furnish their products without being accompanied by any similar disease in the cerebral layer: even in the intense primary inflammation mentioned last, the change which takes place in the cerebral layer, is limited to cloudiness and thickening; the false membrane very rarely produces any adhesion between the two surfaces.

Inflammations of the cerebral layer of the arachnoid membrane present peculiar characters in respect to the condition of the pia mater. We find, on the one hand, that, as arachnitis is usually not fatal of itself, or at least not in an early stage, it sometimes leaves traces of its previous existence, in pretty extensive thickenings of the membrane, in free exudations on its surface, which become converted into circumscribed tendinous patches, or diffused false membranes, &c.; whilst very trifling changes are discoverable in the pia mater, to indicate that an inflammatory process occurred at the same time in it. When, on the other hand, the pia mater is acutely inflamed, and there is profuse exudation into its tissue, the superjacent arachnoid is in no marked degree affected, and its surface is entirely without any free exudation.

The products and consequences of these processes are, considerable thickenings of the cerebral arachnoid, adherent exudations of areolar or dense fibroid texture, which may be smooth, or are granulated like clusters of Pacchionian bodies, insulated shreds of false membrane, or broader and more extended membranes of the same nature, and adhesion of the cerebral arachnoid to its parietal layer. At a later period, plates of bone are sometimes formed in these exudations.

Purulent exudation on the free surface of the arachnoid takes place on the parietal layer, only when the dura mater is very acutely inflamed in consequence of injury of the skull, and caries; and on the cerebral layer, only when a simultaneous acute inflammation of the pia mater also gives rise to an exudation of pus.

It is remarkable that true tubercular exudations do not occur in this membrane.

On whichever layer of the arachnoid these processes take place, that portion of it is exclusively affected which corresponds to the convexity of the hemispheres; and, in proportion as they approach the base of the brain (which they occasionally do), the intensity of the processes, and the quantity of their products, is palpably diminished.

4. *Adventitious Growths.*—Cysts, and lipomatous tumors, are rarely formed in the arachnoid; but both they and the fibroid growths, ascribed to the dura mater (p. 250), when they occur, may sometimes belong rather to the parietal layer of the arachnoid.

a. Besides them, concretions of cholesterine, *cholesteatoma* may be mentioned here. I have repeatedly met with them in the arachnoid at the base of the brain, forming aggregations of delicate white scales, that shine like tendon or asbestos, and are as large as a bean or a hazel-nut, or of still larger accumulations enclosed within epithelial cysts. In one case they were interwoven with extremely fine (microscopic) hair.

b. Fibroid tissue is developed in the diffused and circumscribed opaque thickenings of the cerebral arachnoid; in the instance of the Pacchionian bodies it constitutes a granular form of thickening of the membrane.

c. Bony formations.—Independently of the concretions of bone, which are found in the walls of encysted extravasations of blood, and in fibroid exudations, osseous growths are also developed in this membrane. They are commonly known as ossifications of the dura mater, but in my opinion they appertain rather to the arachnoid. I gather this both from direct examination, and from the fact, that they occur also on the cerebral arachnoid, and on the free layer of the spinal arachnoid. They are almost always situated on the parietal layer of arachnoid lining the dura mater, and much more rarely on its cerebral layer. They occupy the falx usually, the convex part over the hemispheres, and the tentorium very seldom, other parts almost never. Their form is mostly that of plates, as broad as a lentil or a zwanziger[1] piece; their attached surface is smooth and shining, on their free surface they are convex, uneven, and nodulated; their thickness is greatest in the middle, and amounts to two or three lines, their edges are bevelled, irregular, and indented; they are sometimes of a reddish or bluish-red color, sometimes yellowish-white, or white, like a compact bone. They often resemble needles lying singly or joined in groups together; and in this form particularly they are found lying beside the vessels of the falx, or in that part of the dura mater over the hemispheres which adjoins the convex margin of the falx. They may easily be separated from the dura mater; and under the larger plates that membrane appears atrophied.

Bone is rarely formed upon the cerebral layer of the arachnoid; when it is found there, it is almost always over the convexity of the hemispheres, and in the shape of plates, which are smooth on their free surface, and rough on that by which they are attached.

The so-called ossifications of the dura mater are met with mostly in advanced life; their essential importance is far less than that which is usually attributed to them, and they become still more insignificant in proportion as the atrophy, to which the brain is liable at this period of life, increases. As a general rule, they are certainly of moment, when found before the thirtieth year of life. They very commonly coexist with hyperostosis of the cranium, thickening of the vitreous table, especially near the forehead, with adhesions between the dura mater and the skull, cloudiness, and thickening of the inner membranes, &c.

They are composed of true bone, and usually have also a very compact texture.

In some very rare cases bony formations of other kinds are found scattered over various parts of the inner surface of the dura mater, and the opposite layer of the arachnoid. They are crumbling or firm concretions of a reddish, or a white color, and resembling mortar, and are most probably cretified fibrinous exudations.

d. Cancers.—Adventitious growths which belong to the present section, frequently occur on the inner surface of the dura mater; they are quite remarkable for the variety of their external appearance and of their elementary structure. Thus, in regard to the former particular, we meet with delicate villous, vascular, fungous growths, with thoroughly encephaloid formations, with tumors minutely divided into acini, like some glands, with lobulated masses variously streaked with fibres, and so forth.

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[1] [The third part of a florin, and of about the size of an English shilling.—Ed.]

e. Tubercle.—The rare occurrence of exudations to any amount on the free surface of the arachnoid, while they are quite common in the tissue of the pia mater, probably explains why the arachnoid, unlike other serous membranes, should scarcely ever be the seat of tubercular deposit,—why meningeal tuberculosis in every form is restricted to the deposition of tubercle in the tissue of the pia mater.

§ 3. *Anomalies in the Contents of the Arachnoid.*—Some of these anomalies have been already detailed: the accumulation of serous fluid in the arachnoidal sac in any beyond the natural quantity, constitutes another instance of them. When the accumulation is considerable, it constitutes the disease which is known as external, or meningeal, hydrocephalus. Sometimes it is congenital, and is then remarkable for the great quantity of the serum.

It appears in two forms:

a. In that of dropsical sacs, which consist either of arachnoid membrane alone, or, as is more common, of dura mater also, identified with the arachnoid, and attenuated in an extreme degree: they protrude through an aperture in the skull, and form a diverticulum of the arachnoid sac, which communicates with the general cavity by a narrow canal. If, as is very commonly the case, there be no complication with hernia of the brain, such sacs are undoubtedly curable by being emptied and tied.

b. The second is a uniform accumulation of serum in the arachnoid sac, by which the brain is displaced and compressed towards the base of the skull, and the cranium is, at the same time, uniformly enlarged. Although there are some cases, and one in particular in the Vienna Museum, which show that this hydrocephalus may attain very considerable dimensions, yet such an enlargement is extremely rare.

There is an excessive accumulation of serum in the arachnoid sac, which comes on very frequently during extra-uterine life, and calls for some remark.

The normal quantity of serum in children is just sufficient to keep the free surfaces of the arachnoid membrane moist; it may be a few drops, but it does not exceed a drachm; in adults it amounts to three or four drachms. This quantity is found collected in the posterior fossæ of the skull; and if the brain have been carefully removed, it remains in its natural clear and colorless condition: but as in examining and taking out the brain, some blood is usually mixed with it, it appears turbid, and is more or less tinged red.

When there is an excess of this serum, not only has a greater quantity gravitated to the base of the skull, but a part of it also pours out when the dura mater is slit at the mid-height of the brain, along the line where the skull has been divided by the saw. The whole amount may often be estimated at an ounce, an ounce and a half, two ounces, or even more.

The conditions under which considerable quantities are found, are principally two: they may occur,

1. In consequence of frequently recurring habitual congestion of the membranes, or of a varicose state of the vessels of the pia mater.

2. As a result of atrophy of the brain. Under both conditions the excessive accumulation of serum in the arachnoid sac, is combined with hickening of the inner membranes—the cerebral arachnoid, and pia mater,—with infiltration (œdema) of the pia mater in atrophy of the brain, with accumulation of serum in the ventricles, and sometimes with a state of infiltration or œdema of the brain. It is always important to notice that serum is accumulated at the same time beneath the bridge-ike expansions of arachnoid at the base of the brain, especially in the cerebellum; and the more so as the fluid escapes from the latter situa-ion when the brain is removed, and augments the quantity which is found in the arachnoid sac.

This serous effusion into the arachnoid sac, is essentially chronic; but t is subject to sudden or to gradual increase with every attack or augmentation of the congestion, with every advance in the varicose state of the vessels, and in the case of atrophy of the brain, as the vacuum becomes greater within the skull. It is an important question whether there are any acute effusions of this kind which are rapidly fatal; and whether it is possible to recognize them on the dead body as the cause, or as part of the cause, of that form of sudden death which is usually designated "serous apoplexy." That there are such effusions, which may quickly destroy life by paralyzing the brain, can certainly, in the present state of our knowledge, not be denied; but the diagnosis of this mode of death is just as uncertain on the dead body as on the dying patient, for the serum which is found in the arachnoid sac, may be a chronic accumulation that has existed there for a long time, just like that which is contained in the ventricles, or in the tissue of the pia mater, or that which produces œdema of the brain itself. Moreover, the coexistent congestion of the membranes of the brain is usually but slight. And lastly, there are, for the most part, in such cases, various other morbid affections elsewhere, by which the sudden death can be otherwise explained. I shall have occasion to make some further remarks on this subject at another more suitable place.

Not unfrequently the quantity of this serum is uncommonly small, and the surfaces of the arachnoid appear to be without moisture. This is the case in marked enlargement of the brain, especially in hypertrophy.

THE PIA MATER. (THE CHOROID COAT.)

The intimate relation subsisting between the pia mater and the brain, and the frequent coexistence of disease in the latter with that of the former, render the affections of the pia mater those of the greatest importance. In accordance with what has been said of the arachnoid, and in opposition to the general relations of subserous tissue to serous membranes, the most important processes that occur in this situation, viz., those attended with exudation, greatly preponderate in the tissue of the pia mater.

§ 1. *Diseases of Texture.*

1. *Congestion and its consequences:—Hemorrhage.*—There is no

question that congestion of the pia mater (commonly called congestion of the membranes, or inner membranes of the brain) is a very frequent occurrence; and we have already (p. 252) arrived at the same conclusion from our observations on the arachnoid. Yet, on the whole, if we except the "post-mortem" congestion of the pia mater covering the posterior lobes of the cerebrum, any considerable degree of congestion is far less commonly met with in the dead subject than is usually supposed; and there is, perhaps, no respect in which moderation in estimating appearances needs so much to be impressed upon the unpractised observer as in regard to the quantity of blood contained in the vessels of the pia mater: as a general rule, a very moderate injection of these vessels is erroneously looked upon as congestion.

The marked congestions which are met with in the brain and its membranes in very delicate children form an exception to this rule. Much interest attaches to these instances, from their being associated with more or less striking general plethora in children who are usually emaciated.

The congestions are, in general, active, or mechanical, *i. e.* resulting from disease of the heart, or obstruction in the lungs: sometimes they are passive. They are generally combined with a corresponding degree of congestion of the brain; and sometimes they destroy life, either of themselves, as vascular apoplexy, or by causing an effusion of serum into the tissue of the pia mater and substance of the brain.

The terminations and consequences of congestions vary according to the frequency and the duration of their cause. They consist of thickening and condensation (increase of volume) of the pia mater and arachnoid, of permanent infiltration of the former, and a varicose condition of its vessels. Such a state of the inner membranes is well marked after the congestions which are produced by continued and forced exertion of the mind, or by repeated intoxication, especially with alcoholic drinks. Congestions from the latter cause leave behind them an extremely varicose state of the vessels.

The (mechanical) congestions, infiltrations, and thickenings which the pia mater suffers when atrophy of the brain has formed a vacuum within the skull, also require particular notice in this place.

Spontaneous hemorrhages into the tissue of the pia mater (apoplexy of the vascular coat), though they rarely take place in adults, are frequently met with in new-born children and in the delicate period of childhood. In the latter, the part which mostly suffers from hemorrhage is the pia mater at the base of the brain. Cases of this kind must be distinguished from those in which the pia mater is infiltrated with the blood that escapes from an apoplectic spot, whether peripheral or deeply seated. Except in some rare instances, in which a large vessel, or an aneurism of one of the large arteries is ruptured, the source of the bleeding is the fine vessels of the pia mater. Hemorrhages which result from the skull being shattered, or otherwise injured, are mostly accompanied with bruising and hemorrhage on the surface of the brain.

2. *Œdema of the pia mater.*—The infiltration of the tissue of the pia mater with serum, which I have just brought forward as one result of congestion, constitutes œdema of the membrane. It is most commonly chronic: it may be combined with the other changes which have

been enumerated as consequences of repeated and continued congestion; and it may advance till the pia mater measures several lines in thickness, and the serum pours forth in large quantity, when the membrane is cut or torn off. This is especially the case in atrophy of the brain. The infiltered pia mater may be easily separated in large pieces from the brain.

Œdema generally involves the entire pia mater, but it reaches by far its greatest amount over the convexity of the cerebral hemispheres. It may affect merely a very small section of the membrane; and it is thus strictly local when only a part of the brain is atrophied: when the wasting is limited to a few of the convolutions, the œdematous pia mater hangs over them like a loose bag.

In those situations where the cerebral arachnoid is stretched separately and like a bridge over certain parts of the brain, most of the serum is poured out in the free space between it and the pia mater.

The discrimination of an acute œdema, as ever proving the fatal result of an acute congestion of the membranes, is attended with difficulties of precisely the same nature as those which surround the question about the existence of such a disease as serous apoplexy. Œdema of the pia mater is associated with that disease.

3. *Inflammation.*—Inflammation of the pia mater (true meningitis) is the most important of the inflammatory affections of the membranes of the brain. In its essence it is inflammation of a loose areolar tissue. It is impossible to depict its general features without distinguishing two totally different forms of the disease.

a. The first form.

α. Its product is, in general, a yellow or yellowish-green, fibrinous, or purulent exudation into the tissue of the pia mater. This product is effused according to the circumstances of the case, sometimes at separate spots, as between some of the convolutions, or along the course of the larger venous trunks of the pia mater, while the membrane itself is elsewhere infiltrated with an opaque grayish serum: at other times the exudation is made up of flakes of fibrin, and is diluted with a considerable quantity of serum; it is of a grayish-yellow, or variegated greenish color, and pervades the pia mater uniformly; or it may contain but little serum, and be pure fibrin, or pus, diffused in large quantity through the pia mater and its prolongations between the convolutions. In the last case the membrane is manifestly increased in thickness, and may be easily separated from the brain, sometimes without injury, but at other times not without scaling off the surface of the brain with it. It may also be easily torn.

β. In this form the disease usually extends over the convexity of the hemispheres and as it approaches the base of the brain, diminishes in intensity. It rarely occurs at the base of the brain at all.

γ. The individuals who present this form of disease, are in the youthful period, the bloom of life; they are usually strong, at any rate they show no trace of the tubercular dyscrasia.

δ. The disease is usually unaccompanied with (acute) hydrocephalus; at least the exudations found in the ventricles are mostly slight: so also softening of the stomach does not ordinarily result from it.

ε. Except at its periphery, the brain is unaltered by the disease.

This form of meningitis is very frequently a primary and idiopathic disease; at times it is met with frequently, and according to Forget and others, is even an epidemic: and further, it is the usual result of concussion of the brain. Sometimes it is a secondary affection, and as such, it succeeds other inflammations, extensive exudations on serous membranes, for instance, pneumonia, &c., or it is induced by the contiguity of the membrane to circumscribed inflammation going on in the dura mater and cranial bones, by phlebitis of the venous trunks of the pia mater, or of the sinuses of the dura mater by the advance of inflammation or abscess of the brain, and so on; or, lastly, it may be excited by the irritation which adventitious growths of various kinds produce within the cranium.

It usually, as I have already pointed out, spreads over a large artificial area, and is sometimes accompanied with inflammation of the membranes of the spinal cord: even when the inflammation has advanced from some other tissue merely to circumscribed spots of the pia mater, it usually spreads out very rapidly into an extensive (general) meningitis. To this, however, there are occasional exceptions; for inflammation and even suppuration sometimes continue to be limited to their original site; as, for instance, is sometimes the case with the inflammation and suppuration dependent on caries of the skull.

The inflammation is always acute, and more acute in proportion to its intensity, and to the abundance of the exudation. What is called chronic meningitis is really nothing but the various terminations, and the metamorphoses of the products, of an acute inflammation.

Much as it might have been expected, yet are arachnitis on the one hand, and inflammation of the periphery of the brain on the other, far from being constant accompaniments of inflammation of the pia mater. Exudation on the free surface of the arachnoid very rarely occurs, and it is found only when meningitis reaches its most intense degree: the surface of the brain, on the contrary, is certainly more frequently involved. In some rare cases it may be concluded from the terminations and consequences of the inflammation, that it has extended from the pia mater to the surface of the brain in one direction, and in the other to the arachnoid and dura mater, and even to the bones of the skull.

This form of meningitis is frequently fatal; but it often terminates in resolution. When its products change into a cellular or fibroid tissue, the inflammation leaves the pia mater a thickened, whitish, tough, dense membrane; the arachnoid shares in the thickening, the pia mater acquires unnatural connections with the surface of the brain, the cerebral arachnoid adheres to the dura mater, and at length even the dura mater and the skull unite. In some rare cases of this kind, the surface of the brain and skull are bound together by a series of successive normal and false membranes. Such terminations of meningitis are frequently found in mental disease, especially in cases of secondary imbecility.

Meningitis terminates in suppuration only when the inflammation is local, and passes to the pia mater from other contiguous structures.

b. The second form.

a. The products of the second form of inflammation of the pia mater, though commonly mixed with yellow or yellowish-green spots of fibrin, consist in greatest part of an opaline, flocculent, albumino-serous, gelatinous, sero-purulent, and usually very abundant exudation, the color of which is grayish, sometimes shot with yellow or faint green. The opacity of the infiltrated tissue of the pia mater, and of the arachnoid, is palpable in consequence of the transparency of the product. The first-mentioned fibrinous exudation very often assumes the form of granular, plastic nodules, which become tuberculous.

β. It occurs almost exclusively at the base of the brain: and the peculiar product, which was mentioned second, especially accumulates between the hemispheres of the cerebrum on each side, from the optic commissure in front, to the pons, and even over the medulla oblongata behind. In this situation it is deposited amongst the numerous vessels and bands of cellular tissue which pass across the spaces between the pia mater and the bridge-like arachnoid. From thence it may be traced into the fissures of Sylvius, and the longitudinal fissure of the cerebrum, and so on to the convex surface of the hemispheres; for the fibrinous product (that which tends to become tubercle) accumulates along the vascular trunks which run in the fissures, viz.—the arteries and veins of the fissures of Sylvius and corpus callosum, and the latter often appear completely enveloped in the exudation.

γ. From these points the inflammation always extends also to the choroid plexuses and the lining membrane of the ventricles, particularly the lateral ventricles, and there gives rise to the exudation of a similar product, from which a distinct purulent sediment is often deposited: thus it is combined with acute hydrocephalus; and very often it is associated also with softening of the stomach.

δ. The brain is always in a state of (acute) œdema or serous infiltration, and of (hydrocephalic) swelling. (Compare Hydrocephalus, treated of below.) At those parts where the process is most intense, and particularly in the fissures of Sylvius, the cerebral convolutions, especially at their superficial parts, become the seat of red or yellow softening.

ε. The subjects of this form of the disease are mostly children, although it is frequent also at later periods of life. I shall have again to notice that the individuals who are attacked with it are mostly persons of bad constitution, especially of a tubercular dyscrasia, or those in whom tubercle is actually deposited.

This form of meningitis is sometimes primary, sometimes secondary; when attended with tubercular exudation, it is generally secondary, and combined with tuberculosis of other organs. Frequently also, more frequently than the first form, it comes on secondarily as an attendant on various diseases of the brain, such as adventitious growths, particularly tubercle, inflammation, abscess, softening of the brain, and so on, in broken-down constitutions.

The peculiarity of the product of this meningitis appears therefore to arise from exhaustion of the fibrin of the blood by previous exudations of tubercle, or from *hæmatosis*, resulting from the influence of some of the above-named diseases, pre-existing in the brain. Both in its quality, and in the condition on which that quality depends, the product is strikingly analogous to the gelatinous pasty products which are furnished by pneumonia under the like circumstances of a defibrinated condition of the mass of the blood, and are attended with considerable hepatization and extensive tuberculosis.

Though convinced that in distinguishing two forms of inflammation of the pia mater, and in my portraiture of them, I have been perfectly true to nature; yet I must say expressly that the disease presents other exceptional characters.

a. Instances occur, on the one hand, of meningitis at the base of the brain, in which the exudation is fibrinous (plastic), and even purulent;

β. And on the other hand, it is by no means rare for meningitis, on the convex surface of the hemispheres, to furnish a tubercular exudation. Isolated spots of inflammation are then found scattered through the pia mater, especially through the part of it which dips between the convolutions, and it appears infiltrated with a yellow granular exudation. The spots of inflammation are very commonly, too, the seat of hemorrhage, and the membrane adjoining the tubercular mass appears quite filled with coagulated blood. The adjacent surface of the brain is generally in a state of red (inflammatory) softening. These processes are, almost without exception, combined with tuberculosis of other organs, especially with tubercle in the brain; and they appear to be analogous to the lobular pneumoniæ which are attended with tubercular exudation—tubercular infiltration of the lungs.

The meningitis which occupies the base of the brain, very often extends into the neurilemma of the cerebral nerves, as they pass off through the inflamed part.

4. *Adventitious Growths.*—Exclusively of growths, which originate in the brain, and implant themselves secondarily, and in part only, in the pia mater, this class of disease is restricted in the pia mater to tubercle.

Tuberculosis of the Pia Mater.—It is very frequent, and is, of course, one of the most important of all the tuberculoses. It is met with as a chronic disease, but more frequently it assumes the forms of acute tuberculosis, and of meningitis, with tubercular exudation.

a. Chronic Tuberculosis.—In this case the tubercle is found in the form of gray granulations, which, sooner or later, become yellow tubercle. The granules are always grouped or clustered together. They are confined to no particular region. They are constantly combined with tuberculosis of other organs, and they commonly form the starting-point for meningitis and acute tuberculosis of the pia mater. I have never met with complete softening or suppuration of this form of tubercle.

b. Acute Tuberculosis.—Tubercle of this kind appears in the form of the finest granules, scarcely as large as poppy-seed; it may be grayish and opaque, or quite clear and pellucid, like a vesicle. More rarely it assumes the form of larger, miliary, and separate granulations. It needs not merely familiarity with their appearance, but also a close search of the pia mater in order to find them. The congestion to which they owe their origin is often no longer discernible on the dead subject; but they remain always in large numbers, and always occupy a large section, and not unfrequently, indeed, the whole cerebral part of the pia mater: while, together with them, and as the product of the same congestion, a considerable quantity of serous, sero-albuminous, and more or less turbid exudation are found at the base of the brain, within and about the circle of Willis.

The base of the brain is the chief seat of this form of tuberculosis: from thence it extends towards and over the hemispheres; it is rare to find the convex surface of the hemispheres the principal seat of its development. With a little attention the granules may be easily discovered at the base of the brain by removing the bridge of arachnoid, but it requires the closest search to detect them in the pia mater, covering the convexity of the hemispheres: in this situation they are clustered in the intervals between the convolutions, and are very often further concealed by a good deal of congestion of the membrane.

The amount of the accompanying (acute) hydrocephalus and œdema of the cerebral substance bears a direct relation to the extent to which this form of tuberculosis is developed. It is most frequent in children and young persons, but it may occur at any period of life. It is very rarely the primary disease, but almost always depends upon some previous tuberculosis, either chronic tuberculosis of the pia mater, tubercle of the brain, or the like disease of the lymphatic system, or lungs. Not unfrequently it forms part of a general acute tuberculosis which has attacked the most different organs and tissues, either all at once, or quickly one after another.

c. Meningitis, with tubercular exudation, so called *tubercular meningitis.* This is the second form of meningitis with a fibrinous, yellow, granular product, which was described at p. 262. Sometimes it is combined with an acute exudation of gray granular tubercle (acute tuberculosis). Sometimes also it appears in the form of isolated spots of inflammation of the pia mater covering the convex surface of the hemispheres (described at p. 264). In both forms it has the same combinations and starting-point, as the acute tuberculosis.

It is remarkable that the pia mater of the cerebellum is very rarely the seat of tubercle.

On one occasion I met with a tumor in the pia mater consisting of a cavernous tissue; it was a specimen of teleangiectasis (splenic nævus, Aftermilz), and was situated at the upper part of the left cerebral hemisphere.

OF THE PROLONGATIONS OF THE ARACHNOID AND PIA MATER WITHIN THE BRAIN.

§ 1. *Diseases of the Choroid Plexuses.*—The choroid plexuses are subject to congestion, opacity, and thickening, and to a varicose state of their vessels: and these changes are mostly observed when the pia mater and arachnoid are in a similar condition. The choroid plexuses share more or less also in the processes of exudation, which arise from meningitis and acute hydrocephalus, and are then opaque, swollen, infiltrated, and covered with flocculent pseudo-membranous fibrin, or with purulent exudation.

Cysts and *calcareous formations* are the principal adventitious products in the choroid plexuses, and they are frequently met with.

The cysts, vesicles, or, as they are sometimes named, hydatids, of the choroid plexuses, are frequent and well known. Various misconceptions have prevailed as to their nature and cause, and undue importance has been attached to their presence. They have been taken for dilated lymphatic vessels, for enlarged capillary vessels (or terminations of arteries), and for new growths; and their value, in the scale of post-mortem appearances, has been commonly over-estimated. They form bladders, sometimes with very thin, sometimes with pretty thick, vascular walls; they may be loose and pendulous, or filled and tense; they vary in size, equalling a poppy-seed, millet-grain, or bean; and when very numerous, they give the choroid plexus the appearance of a bunch of grapes. They occur only in the lateral ventricles, at least none that occur elsewhere are large enough to be detected in making the post-mortem examination with an ordinary amount of care, that is to say, none above the size of millet-seed. They occupy the convex portion of the plexus, especially towards the posterior cornu of the ventricle. They appear to me to be a disorder of the gland-like acini and villous appendages of the choroid plexuses, and, therefore, to bear a close analogy to the cysts, which are so often developed from the Malpighian bodies of the kidneys, especially in consequence of inflammation and Bright's disease.

In young persons they are almost constantly absent; but they are very frequent in those who are advanced in life, and in whom there is hydrocephalus arising from vacuum, or thickening and œdema of the inner membranes of the brain, &c.

These cysts, for the most part, have but one chamber, but they are often divided into compartments by delicate partitions. They generally contain a clear serum, but the fluid they enclose is often turbid, whitish, and like lime-water, and at last there is often whitish or yellowish *bone-sand* in them besides. These concretions incrust the cells, and are analogous to the sand met with in the brain; they are found, too, when there are no cysts, in the texture of the villi of the choroid plexuses.

Tuberculosis.—This is an extremely rare occurrence: even when there is an exuberant production of tubercle in the tissue of the pia mater of the base of the brain, there is very rarely any trace of it in the adjoining choroid plexus.

Cancer.—This disease, occurring alone in the choroid plexuses, is one of the very rarest ever met with. I have seen a medullary degeneration of the choroid plexus of the fourth ventricle.

§ 2. *Of the Lining Membrane of the Ventricles.*—The internal membrane of the ventricles of the brain is composed of a very delicate continuation of the arachnoid and pia mater, and a layer of epithelium. The most frequent and most important diseases to which it is liable, have, from one most striking characteristic which they present, viz., an excessive accumulation of cerebro-spinal fluid, been included together under the title of hydrocephalus.

Although it is true that the presence of an excessive quantity of fluid in the ventricles, and structural disease of their lining membrane, are not essentially characteristic of all cases of hydrocephalus, but are sometimes secondary, and occasioned by disease of the brain, and therefore that all the forms of hydrocephalus do not rightly belong to the present section, yet I prefer treating of them altogether, for two reasons:

(1.) Because the most important of all the forms, viz., acute hydrocephalus, arises from disease of the lining membrane exclusively; and

(2.) Because, in spite of their differences, it has become a prevalent custom to associate them together, both in thought and in description.

Though, for the sake of convenience, I thus disarrange the subject, it will be restored again by referring to each of the forms I am about to describe in the place to which it properly belongs.

1. *Hydrocephalus.*—I would here offer the following general remarks: that, in accordance with what has been already said, by the term hydrocephalus is meant "Hydrocephalus stricte sic dictus internus,"—dropsy of the ventricles: and that I retain the usual division into acute and chronic hydrocephalus, as it appears to be that which still possesses the most practical value. The chief seat of hydrocephalus is generally the lateral ventricles, and they are for the most part symmetrically affected. Exceptions will be pointed out in their places.

A. *Acute hydrocephalus.*—This is both the most frequent and the most important of the forms of hydrocephalus—the acute dropsy of the ventricles. Anatomy discloses two essentially different forms of it.

a. The *first form.* Its anatomical characters are—

α. The effusion of a fluid which is thin or somewhat thick, of a grayish color, or grayish-yellow tinged with green, and more or less turbid, in proportion to the quantity it contains of plastic matters capable of assuming some of the primary forms of organization. It is very often found to have separated into two parts—one fluid, and the other of more consistent and deeper color: it has, in fact, become clear from certain of its elements having fallen to the bottom as a sediment. These elements are usually found in the most dependent part of the lateral ventricles; viz., their posterior cornu. On minute examination of the effusion, its opacity is found to arise from the presence of the elements of a plastic exudation, nucleoli, nuclei, cells at various stages of development, and true pus-cells; and of them the sediment, distinguished by the deeper yellowish or greenish color which it presents, is, for the most part, composed. But cast-off epithelium in course of solution, shreds of the lining membrane of the ventricles, and even shreds of nerve-tubes, are also found in the effusion, and all contribute to its opacity.

In some few cases a more solid exudation is observed besides the fluid effusion; it adheres here and there, especially on the corpora striata and optic thalami, in membranous plastic flakes to the lining membrane.

β. The quantity of the morbid effusion cannot be accurately determined, because of our uncertainty as to the pre-existing or normal quantity of fluid in any particular case. The whole quantity, however, inclusive of that which previously existed, is generally not considerable, and usually does not exceed an ounce; oftentimes it is scarcely half so much. On the other hand, in a few cases in which acute hydrocephalus supervenes upon chronic (whether the latter be congenital, or have come on early in life), the quantity of opaque contents of the ventricle is very considerable, and the greater part of it appears to have been produced by the recent process. The enlargement of the ventricles corresponds to the quantity of their contents, and in ordinary cases is but slight.

γ. The lining membrane of the ventricles becomes opaque, soft, and dissolved, and shreds of it consequently appear in the effusion.

The choroid plexuses become opaque and softened, and are very commonly enveloped in a villous and slightly shreddy layer of grayish, or grayish-yellow exudation.

δ. From this point further changes extend in two different directions, to the cerebral substance, and to the inner membranes at the base of the brain. The affection of the membranes is the more essential part with respect to the nature of the process.

(1.) In the neighborhood of the ventricles the cerebral substance is percolated with serous fluid—infiltrated—to such a degree, that it seems as it were in a state of watery softening; very often, too, it is streaked or dotted with ecchymoses. The cerebral substance is thus affected wherever it adjoins the lining membrane of the ventricles; but very commonly the fornix and septum are softened to the greatest degree, and the latter is sometimes quite broken down and perforated. From this spot the œdema extends through the whole of the cerebral hemispheres, always, however, diminishing gradually as its distance from the ventricles increases, and always being greatest close to them. Hence the cerebrum swells, and increases in actual volume; its convolutions are forced against the walls of the cranium, and flattened; and in the same manner the cerebellum and pons are depressed and flattened in a marked degree. The inner membranes covering the convex surface of the hemispheres, being involved in the pressure against the cranium, appear bloodless. The cerebral substance also is bloodless and pale; it has a singular dull white appearance, and a peculiar soft and doughy consistence arising from its uniform moistness.

(2.) With the affection of the lining membrane of the ventricles and choroid plexuses, the diseased condition of the inner membranes at the base of the brain, forms one entire disease, not only by continuity, but in essence also. It takes the form of meningitis, especially of that described at page 262 as the second form of meningitis; or else it appears as acute tuberculosis of the pia mater at the base of the brain. In speaking of the serious character of these processes, I have already grounded it on their extension to the lining of the ventricles, that is, to their combination with acute hydrocephalus.

b. The *second form*. Its anatomical characters.

a. Effusion of a clear, colorless, serous fluid into the ventricles; sometimes it is slightly turbid, from being mixed with shreds of the lining membrane and of cerebral matter.

β. The effusion varies in quantity, being sometimes slight, but more frequently considerable, amounting even to six ounces.

γ. The cerebral substance around the ventricles is generally in a state of watery softening, in which the rest of the cerebral mass shares, only in a less degree, the change in it not exceeding ordinary œdema. The whole brain presents the same swelling and the same general condition as have been described of the first form, but generally, even to a more marked extent. The cerebral substance surrounding the ventricles and the lining membrane, may sometimes be found in the dead subject in a normal or nearly normal condition; but this happens only in some extremely rare cases, in which the disease is known to have run an acute course.

The most remarkable, and one of the most important of the post-mortem appearances, in both the principal forms of acute hydrocephalus, is this almost constant softening, or, as it is called, maceration of the cerebral substance about the ventricles. Before proceeding to any general remarks, some notice of the nature and import of this appearance is indispensable.

In so far as regards anatomical disorganization, I hold it to be in itself no very essential part of the disease; it is, however, certainly very important, and perhaps even more so than the effusion into the ventricle itself. For first, it involves cerebral substance, and secondly, it attacks that substance in a very acute manner, and rapidly produces disorganization of it. It is, in fact, nothing more than an acute œdema of the highly delicate and easily injured texture of the brain, and the equally delicate lining membrane of the ventricles; but so rapid, occasionally, is its progress, and to such degrees does it advance, that it gives rise, for the most part, to countless lesions of continuity, and thus, in the form of softening, disorganizes the brain and destroys life: if it should advance more slowly, or to a less degree, it may very often continue a long time without marked symptoms.

The mode of origin, and the import of this œdema, will be more distinctly understood from the following particulars:

(1.) It corresponds entirely to the œdema which surrounds every spot of inflammation, and to that which ensues upon acute congestions.

(2.) And further, it is in my opinion worthy of remark, that if an effusion takes place so rapidly that room cannot at once be obtained for it in the ventricles, by displacement of the brain, the resistance from within is so great as to hold, or press back, the exudation, and a portion of that which should be exuded from the lining membrane of the ventricle, is poured into its tissue, and into the adjoining part of the brain. The greater the intensity of the process, and the quantity of its products, the sooner does infiltration ensue, and break down the textures; and it will the more readily take place, if the brain have been œdematous before, or the cerebral mass around the ventricles have been distended by a pre-existing effusion.

(3.) In the first of the two forms of hydrocephalus, especially in that with which true meningitis is combined, the serous exudation which gives rise to softening of the cerebral substance around the ventricles is sure to contain a portion of coagulable or plastic materials, capable of assuming a primary organic form; in the second it is entirely, or almost entirely, composed of pure serum. This accounts for the circumstance that in many cases, the macerated cerebral substance, when minutely examined, is found to contain the so-called exudation-corpuscles, exudation-cells, nucleated and primary cells (pus-cells), while in other cases these are entirely wanting. (Gluge.)

This state of the brain, then, may be suitably classed, as has been done already, with the termination in softening, and may be named white softening, hydrocephalic softening. I shall have some further remarks to offer upon it when treating of œdema, especially in the article of Softening of the Brain.

This white softening of the cerebral substance is sometimes accompanied with yellow softening, more particularly when the case is one of the first form of hydrocephalus, and combined with meningitis.

Moreover, I have alluded above to the softened cerebral substance being sprinkled or streaked, as it were, with red ecchymoses: both forms of this disease present this feature, but it is more common in the first form. It arises from the laceration of the delicate vessels, which are torn when the cerebral texture is broken down; but there is very often far less of it than the degree of disorganization would lead us to expect. The question which this suggests, admits as yet of no other solution than that the simultaneous swelling of the whole brain so obstructs, and precludes the injection of, the cerebral vessels, that those which are torn are empty.

With respect to the nature of acute hydrocephalus, an inquiry which has led to so much discussion, that of the *first form* of the disease is perfectly clear: it is either an extension of meningitis of the base to the ependyma of the ventricles,—of a meningitis attended with an exudation that contains less than the average of plastic material, that bears traces of a faulty constitution of the blood, and that, in its fibrinous portion, is very often tuberculous; or it consists of a supplemental, and, for the most part, serous exudation, accompanying an acute deposition of tubercle in the pia mater, at the base of the brain. This form of acute hydrocephalus, therefore, is either actual inflammation, or an exudative process having a general connection with it. Although I cannot coincide in the opinion of several French observers, who think that acute hydrocephalus is never anything but meningitis,—by which term acute tuberculosis is also meant,—yet I so far agree with them as to believe, that in the great majority of cases such is the fact, and that the meningitis is very commonly of a tuberculous character.

This form of hydrocephalus occurs both as a primary and substantive, and as a secondary affection. When secondary, it attends the diseases of the brain which have been mentioned already, at p. 263; viz., inflammation, abscess, and a yellow softening, adventitious growths within the skull in general, but more than all, with tubercle of the brain: with that disease it associates itself, either in the form of meningitis with tuberculous exudation, or in that of acute tuberculosis. It is very frequently the means by which those diseases of the brain destroy life. The hydrocephalus which originates with tubercular meningitis, or with acute tuberculosis, is very rarely a primary disease, but supervenes upon some previously existing tuberculosis, upon that in the brain particularly, as well as that in the glands or in the lungs: when connected with acute tuberculosis, it forms one of the many local parts of the general disease. (Compare p. 265.)

The subjects of it are, for the most part, children; but adults, and even persons advanced in life, suffer from the secondary form, especially when it is a process connected with tubercle.

The *second form*, though an acute hydrocephalus, cannot be admitted to be inflammatory: it bears, in fact, none of the characters which mark inflammatory states or products. It arises from congestions of various kinds: such as are connected with the development of the brain in childhood, or those produced by chronic eruptions on the scalp, by the irritation of morbid growths within the skull, &c. These congestions are analogous to those from which acute dropsies of many of the serous and synovial membranes result,—dropsy, for instance, of the tunica vaginalis testis, and the acute œdemas. It may be occasioned, too, by the congestions which follow concussion of the brain, or mechanical obstructions, such as disease of the heart, rickets of the thorax, impermeability of the lungs in tuberculosis and phthisis, chronic catarrh of the bronchi, chronic pneumonia, &c. The result of these congestions is an excessive effusion of serum, first from an apparatus especially adapted for that purpose, viz. the lining membrane of the ventricles, and then into the brain itself.

These effusions, if the process which gives rise to them be very intense, destroy life at once, upon their first occurrence; otherwise they are fatal only after being several times repeated. Hence it is that the quantity of fluid found in the ventricle varies so much: the larger accumulations, those which amount to as much as six ounces, are apparently the sum of several smaller effusions, occasionally repeated. The enlargement of the ventricles corresponds to the quantity of serum within them; and the skull enlarges in an equal degree until the sutures are closed.

This form of hydrocephalus, then, not unfrequently runs a protracted, subacute course; and it tends the more to do so, in proportion as the several exudative processes are slight in degree, and as the skull retains more of its early elasticity. And further, the less distinguishable the several exudations are from one another, the more this form is allied to chronic hydrocephalus.

It is sometimes a primary and substantive disease, sometimes a secondary. When primary, it is in childhood, like chronic hydrocephalus, remarkable for its combinations, of which I have to speak hereafter; and for being intimately connected with a deeply-lodged anomaly of the general vegetative processes. When secondary, it is frequently occasioned by various diseases of the brain, as inflammation, abscess, and by morbid growths in the skull.

Considered apart from chronic hydrocephalus, which stands in close proximity to its subacute variety, it is certainly, on the whole, more rare in childhood than the first form; but then it may occur much earlier, being met with in the first year of life, and doubtless, also, in the fœtus. Moreover, it is not very rare at any later period of life, up to old age, for it is occasioned by mechanical congestions in the course of various chronic adynamic diseases, which are attended with a dropsical crasis of the blood.

Both forms of acute hydrocephalus are attended by certain combinations, some of which are common to both, while others are peculiar to one of them. They are partly constant and essential, and partly neither constant nor essential.

The first form is, in the great majority of cases, combined with a tuberculous diathesis, and the local tuberculoses, mentioned at p. 265: these constitute the fundamental anomaly.

The second form combines with it several abnormal conditions, especially in children; and very often all the disorders enumerated below occur together, and form one complex morbid state, that manifests a thoroughly depraved working of the vegetative process. They are—

(1.) Hypertrophy of the whole system of lymphatic glands, and of the follicular apparatus of the intestinal mucous membrane.

(2.) Arrested decay or involution of the thymus gland.

(3.) Chronic catarrhs, especially of the bronchi.

(4.) Rickets and its attendants.

One coincidence, which deserves special attention amongst these combinations, is that of hydrocephalus with hypertrophy of the brain. The latter is well known to be very commonly associated with general rickets; but the former is so constantly found to be connected with rickets of the thorax, that Engel has given the name of hydrocephalic to that particular distortion of the chest.

A very common and essential combination with both forms of the disease, and one with which the fatal result is frequently connected, is softening of the stomach.

The following are unessential, and, to a certain extent, merely accidental combinations: hypostatic congestions of the lungs, lobular pneumonias, slight pleuritic exudations, enlargement of the liver, &c. Intussusceptions are very frequently met with in the intestines: but, although Abercrombie attributes them to the same cause as the vomiting which occurs in the course of the disease, they have certainly not existed for any length of time, and must rather have arisen during the agony; for they present no trace of any congestion or swelling of the bowels from the strangulation of those vessels of the mesentery which are invaginated with the bowel.

Terminations.—The great fatality of acute hydrocephalus is well known, and may be accounted for. The first of the two forms, when a tubercular process, is undoubtedly always fatal; but when it is a simple meningitis, it may, as well as the second form, be outlived and cured, provided it be moderate in degree, and especially if the brain have escaped complete destruction by white softening.

This termination, in a more or less complete cure, may be reduced to the following particulars:

(1.) The products of the process may be entirely reabsorbed, and the brain be restored to its natural consistence, size, and figure.

(2.) A part of the effused fluid, or the whole of it, may remain in the ventricles, and both they and the skull may be permanently enlarged. This can only be conceived to take place in the child just before the skull is completely ossified. The acute hydrocephalus thus becomes a chronic accumulation, which is capable even of further gradual increase.

(3.) In that case, the lining membrane of the ventricles very often remains thickened in various forms and degrees. The quantity and density of its tissue are increased, and the plastic exudations remaining upon its surfaces become converted into a cellular or fibroid tissue, and covered with a layer of tesselated epithelium; thus both contribute to the thickening of the wall of the ventricle. The new tissue assumes various forms, similar to the false membranes which occur on serous surfaces.

As its presence on so delicate a substratum as the ependyma is of some interest, and as it may be the means of leading to a more accurate examination of the cases in question, I will give a more detailed description of it.

α. The lining membrane sometimes appears covered with a granular film, like the finest sand, which has a transparent crystalline, or an opaque, grayish-white appearance, and can be detected only by looking carefully while the light falls favorably upon it. It may occasionally be seen at every part of the lateral, third or fourth ventricles, but it is generally most developed at particular spots, as the corpus striatum, and tænia semicircularis, and especially in the anterior cornu of the lateral ventricle.

β. More rarely it forms coarser granulations, which are then more prominent, and in time become nodules attached by a pedicle. These granulations, more particularly, are analogous to the false growths of the same kind, which occur on other serous membranes, and to the Pacchionian bodies on the arachnoid.

γ. Sometimes the new tissue is smooth, membranous, and superficially attached, and forms separate, round, white, opaque, islands, or "plaques," which are not unfrequently thinner in their middle, and, as it were, perforated (gefenstert, latticed): this form is analogous to the tendinous spots.

δ. At other times the tissue is similar in its character, but instead of forming separate islands, it is continuous, and the whole seems knitted or areolar, and forms an adherent network of false membrane, which may generally be easily raised from the surface.

ε. Or, lastly, it forms false membranes of considerable and nearly uniform thickness, which are for the most part, intimately united with the lining membrane.

In these last sometimes bony concretions are developed.

In some very rare cases of chronic hydrocephalus in children, especially of congenital but advancing hydrocephalus, in which, also, the thickenings just described exist, a peculiar appearance is met with on the walls of the ventricle. The cerebral substance protrudes into the ventricle at various spots, probably those where the ependyma is relatively thinner, and forms rounded, smooth bosses, with broad bases, as large as hempseed or peas. I have had two opportunities of observing this peculiarity.

(4.) Does it happen, as Otto, I believe, first asserted, that hydrocephalus (of course I mean the second form, that which is allied to chronic hydrocephalus), is ever cured by the supervention of hypertrophy of the brain? I have already (p. 272) considered the combination of hydrocephalus and hypertrophy of the brain, which is occasioned by rickets: I believe, further, that the hydrocephalus (the hydrocephalic process) may itself sometimes give the first impulse to hypertrophy of the brain: but that any compensation for, or cure of, hydrocephalus is effected by hypertrophy, appears to be altogether problematical. Such an opinion is founded upon the fact, that in some large skulls, of hydrocephalic shape, the brain exceeds the normal size and weight. But, I believe, that these are cases in which the hypertrophy having taken place in childhood, has continued ever since; and that belief is confirmed by the resemblance, in shape, which subsists between the skull in hypertrophy and the hydrocephalic skull, as well as by the difficulty which the similarity in the symptoms of hypertrophy and hydrocephalus imposes, upon our determining positively what disease of the brain did exist in childhood. So far as I am aware, the morbid increase in the volume of the brain in hydrocephalus, as well as its normal growth, takes place always in the neighborhood of the enlarged ventricles; it is a peripheral deposition around them: and the skull goes on increasing in size to whatever extent its closure may be prevented by the hydrocephalus.

B. *Chronic hydrocephalus* may be subdivided into congenital hydrocephalus, and that which commences at various periods of extra-uterine life: besides these, there is a third and entirely different form, hydrocephalus ex vacuo. The distinction between the first two forms is not made by any essential difference between them, for in the most important particular, viz., their cause, they are undoubtedly alike; but congenital hydrocephalus presents such very marked peculiarities that the distinction appears justifiable.

The general anatomical characters of chronic hydrocephalus are a large accumulation in the ventricles of clear and colorless serum, which contains very little animal matter, and a thickening and toughness of their lining membrane, for the most part to a considerable degree.

a. Chronic hydrocephalus, commencing after birth.—This is either a termination or continuation of acute hydrocephalus, especially of the second form of it; or else it is chronic from the first. The symptoms during life in the latter case were not such as to indicate the existence of any acute disease of the brain; they were rather those of a long-continued disease which occasionally underwent exacerbations.

This form of hydrocephalus may occur at any period of life; but it is most frequent in the first years, and then attains its most advanced degree. The quantity of serum accumulated varies considerably, and depends upon the duration of the disease, and especially on the circumstances of the skull being closed, or not, when it commenced, and whether its origin date from early childhood. In the first case it amounts to two or four ounces; while in children, and in adults who have had the disease since childhood, it may be as much as six, eight, or ten ounces, or even more. In children the skull increases in volume in proportion to the quantity of serum and the enlargement of the ventricles, and, at the same time, acquires the well-known hydrocephalic form, which so often continues throughout the remainder of life. This hydrocephalus then precisely resembles the congenital disease, and might in any case be taken for it, were there no certainty that it had come on since birth. The anatomical description of it agrees entirely with that of the congenital disease.

In respect to its causes, and the mode of its development, it essentially corresponds with the second form of acute hydrocephalus; oftentimes it is a primary and substantive disease, but very frequently it is secondary and symptomatic. Thus in the child as well as in the adult it arises as a primary disease from repeated and continued active congestions, such as, in the former, occur during growth, and in the latter, more frequently from excessive exertions of mind, repeated intoxication, &c. As a secony affection, it is a consequence of different diseases of the brain, par- larly of adventitious growths within the cranium; or it comes on in course of chronic diseases of the lungs, &c. Some growths are so ated that their pressure renders a sinus, particularly the straight ıs, impervious, and thus gives an especial occasion to chronic hydro- halus (Barrier).

Vhat has been said of the combinations of the second form of acute lrocephalus applies to the chronic disease also. When thus combined uns a lingering course, and has the character of a constitutional affec- ı.

Terminations.—*a.* Two circumstances may interfere with a complete e of the disease by the reabsorption of the fluid, the great quantity umulated, and the extent to which the skull is correspondingly en- ged. The only real cure is a cessation of the process, and then the ntity of this fluid continues undiminished throughout life. The ques- ı, as to a cure being brought about by means of hypertrophy of the in, has been already met at p. 273.

. The disease may terminate fatally—

1.) By pressure, and consequent palsy of the brain, after having ched a certain degree, and having sometimes, in adults, occasioned orption of the inner table of the skull;

2.) By the supervention of, or advance of the process to, a consider- e acute exudation into the substance of the brain, by œdema of the in, and hydrocephalic softening;

3.) By an attack of acute hydrocephalus and meningitis.

. *Congenital hydrocephalus.*—This form of hydrocephalus is one of eminently chronic character; it exists at birth, and usually has then eady made considerable progress; but, if not, it soon increases, and, the extraordinary size which it attains, and the amount of deformity roduces, it constitutes the most striking example of the disease.

It is then distinguished by the large quantity of serum which the ven- cles contain, and by the extent to which they and the skull are en- ged. Those cases in which no monstrosity of the brain coexists, may portrayed as follows: The quantity of serum contained in the ven- cles amounts to several pounds, 6–10, or even more: the ventricles expanded into large elliptical cavities, or membranous sacs; and ir ependyma or lining membrane is generally much thickened. The ebral mass around the ventricles, especially towards the top of the ad, is attenuated, and sometimes measures scarcely a line in thickness: nay be even so reduced as to be but a just perceptible layer covering membrane. In one case, which is preserved in the Vienna Museum, s broken quite through, at the upper part of the hemispheres, by the ckened membranous walls of the ventricles, and has receded from m to a considerable extent. Internally and inferiorly, the serum by pressure flattens the corpora striata and optic thalami, and passing o the third ventricle, it forces those bodies asunder also; the corpora adrigemina become smoothed, the commissures stretched, and the gray nmissure very commonly wasted; the pillars of the fornix are forced rt, and, with the septum, driven up against the corpus collosum; y are also either all much raised, or the septum is enlarged, very much thinner than natural, and broken through in one or more places of various size. The floor of the third ventricle is thin and transparent, the cerebellum is flattened from above: the pons is flat and spread abroad; the crura cerebri are separated; the pituitary gland is flat, or even concave, and wasted from pressure. The size of the cerebrum is greatly disproportioned to that of the cerebellum, the parts at the base of the brain, and the nerves. The surface of the cerebrum is flat, its convolutions are but just indicated, and could not be recognized; and all the membranes of the brain are unusually delicate and thin. The head is quite remarkable for its size and its deformity. (Compare p. 168.)

Congenital hydrocephalus is far from constantly agreeing with this picture: on the one hand, the quantity of serum, and the enlargement of the ventricles and skull, may be less than has been stated, and may indeed only just exceed the normal standard: while, on the other hand, under certain conditions, the development of the brain may be faulty, even to monstrosity.

The mode of origin, or pathogenesis, of congenital hydrocephalus differs most probably in no essential particular from that of the chronic hydrocephalus which commences in the extra-uterine periods of life. It may come on in the fœtus as acute hydrocephalus, or appear originally in the chronic form. The general arrangement of the skull of the fœtus, and the manner in which the cerebrum itself is developed, are both highly favorable to an excessive accumulation of serum. And I believe, that the really essential part of congenital hydrocephalus, that which arrests the development of the brain, is the affection of the ependyma; that, in proportion to the degree to which the hydrocephalus has advanced, and according to the period of fœtal life at which it commenced, it does, in various manner, and to different extent, arrest the development of the brain, and occasion monstrosity of it; and so far contains the ground of its alliance with hemicephalus, hydrencephalocele, singleness of the cerebrum (cyclopia), &c.

What has been said of the combinations of acquired chronic hydrocephalus, describes those of the congenital disease also. A congenital dwarfish growth sometimes takes the place of rickets.

Terminations.—The hydrocephalus of the fœtus, even when it has reached a considerable extent, is not unfrequently inherited by the child, the youth, and even the adult. During the intervals in which the disease is quiescent, the brain grows, and acquires its normal volume, and the skull, continually advancing over it, at length closes. In some rare cases, growth passes beyond its normal bounds into hypertrophy; but with regard to the cure of hydrocephalus by hypertrophy, what has been said at p. 273, may be applied in this place.

The disease sometimes proves fatal by the pressure which the continual accumulation of water exerts upon the brain. Frequently, too, acute inflammation of the ependyma, and meningitis arise in its course. I have, moreover, seen it terminate by rupture of the brain and dura mater, and extravasation of the serum of the ventricle beneath the pericranium and adjoining aponeurosis.

Finally, considerable importance attaches to the hemorrhages which take place in the course of the disease: they are met with both in the arachnoid sac and also, and more especially, in the dilated ventricle. They are remarkable for the length of time during which they are borne, as is attested by the metamorphoses of the extravasation. The way is most probably prepared for their occurrence by the stretching of the vessels of the membranes that cover the brain and line the ventricles, during the distension of the latter; and it is by the final rupture of those vessels that they are actually produced.

c. Hydrocephalus occasioned by a vacuum within the skull.—When an empty space is formed within the skull by a reduction of the volume of the brain, it is filled up (as already pointed out, pp. 253 and 260), by an increase of the volume of the inner membranes of the brain, and especially by an extraordinary exhalation of serum into the tissue of the pia mater, the sac of the arachnoid, and the internal cavities of the brain, more particularly the lateral ventricles. These changes result from the congestion of the vessels which the vacuum produces. The most common instance of effusions of this kind into the ventricles, is that which occurs when the brain is atrophied in old age; a condition which has obtained the title of hydrocephalus senilis. From the process of involution of the brain, which gives rise to the affection in this instance, being so free from complication (einfach), from its uniform occurrence in both halves of the brain, and, lastly, from its frequency, it may serve as an example of this species of hydrocephalus. But the same condition is met with also in all cases of premature senility of the brain (senium præcox cerebri) and in every spontaneous and primary, as well as in every consecutive atrophy. Examples of the latter kind occur after recovery from repeated attacks of apoplexy, after inflammation of the substance, which has terminated with induration and wasting of the diseased portion, after the closure of an abscess, the healing of a wound attended with loss of substance, &c.

Hydrocephalus is symmetrical, or otherwise, according to circumstances. The quantity of serum contained in the ventricles, and the dimensions to which these cavities are enlarged, are measured by the degree to which the brain is atrophied: the former very commonly equals an ounce and a half, it often amounts to 2 or 4, and may reach even 6 ounces, or more. As the quantity of serum increases, it accumulates in greater proportion in the dilated third ventricle, and produces especial attenuation of the gray commissure, the pillars of the fornix, and the septum ventriculorum: the septum may even be more or less perforated.

The serum in the ventricles, like that contained in the sac of the arachnoid, and that infiltrated through the tissue of the pia mater, is remarkably clear.

As the membranes at the periphery of the brain increase in volume, so also does the lining membrane of the ventricles become thicker than natural; and it often bears on its surface some analogue of the Pacchionian corpuscles of the arachnoid; for the membrane is covered over with fine granules, which are either clear like crystals, or opaque and white. This thickening is the principal cause of the resistance which is experienced in slitting up the walls of the ventricles.

Serous cysts on the choroid plexuses are frequently found in hydrocephalus senilis.

Again, the brain may be generally of small size, though it exists in all its parts; and the skull is diminished to a corresponding extent: but of this state, microcephalus, as well as of several other instances of deficient development of the brain, a further account will be given amongst the anomalies in its form.

There is, besides, one instance of congenital deficiency of more or less of the brain, which I have not put in the same series with those already mentioned, because, to say the least, it is highly probable that it owes its origin to an attack of hydrocephalus at some period (generally a very early period) of fœtal life. It is the instance known as anencephalus, hemicephalus, and also as acrania.

There is much difference in the extent to which different cases of hemicephalus proceed, depending partly on the extent of the previous hydrocephalus, but principally upon the period of fœtal life at which the disease of the brain commenced. Sometimes that organ is wholly wanting, and only the membranes are found at the base of the skull, with the cerebral nerves sunk into them: sometimes a few rudiments of the brain exist, particularly those structures which compose its base; while it is covered with a membrane, formed of much attenuated skin, and dura mater, which exhibits traces, more or less distinct, of having been ruptured. The vessels of the inner membranes are generally numerous and gorged, the membranes themselves are filled with extravasated blood, they present a honeycombed arrangement of their structure (which has been compared to hydatids), and contain some grayish-red cerebral substance. The brain itself is unusually vascular and soft, and appears as if it had been macerated. The roof of the skull is almost entirely wanting; for the usually expanded frontal and parietal bones form mere small and slender streaks, or irregular triangular plates of bone, and are sunk down upon the base: and the broad occipital plate is shrunk to a few rudiments, or severed by wide fissures. A vault may be formed to the skull by these rudiments of the bones, but it is very low, and divided by wide fissures from before backwards. The bones at the base, if they are not divided also, are small like the occipital bone, but very thick and coarse.

In other cases, only a small part of the cerebrum is destroyed; and, as the greater part of the brain remains, the cavity of the cranium is proportionally capacious, and the deficiency of bone is confined to its uppermost part.

Hemicephalus is allied to a certain stage of hydrocephalus. Within a skull of normal size or enlarged, but which is closed, there exist no cerebral hemispheres, but a sac, surrounded with the cerebral membranes, and filled with serum, while the base of the brain lies at the lower part of the skull, more or less rudimentary and misshapen.

Such a case exhibits clearly the alliance which subsists between hemicephalus and hydrocephalus, and the foundation of the former in the latter; but the combination of hemicephalus with encephalocele, that is, its origin from hydrencephalocele, exhibits it more clearly still. In this instance of hemicephalus, a part or even the whole of the brain, destroyed in the manner above described, lies outside the skull; the cranial vault is split along the mesial line by a greater or less fissure, and is low in every case, but it is sometimes quite sunken to the base.

ut further, we have an opportunity sometimes of demonstrating this se of hemicephalus, by direct observation at the time of its occur- e. The skull of a fœtus, at such a time, is found distended and rocephalic, and at the vertex a slough is seen, produced by the sure and stretching.

astly, hemicephalus is very frequently combined with the same in- ces of arrested development as the higher degrees of congenital rocephalus. It is often accompanied, too, by fissure of the vertebral mn (spina bifida): when the occipital bone is split completely through, cervical vertebra are nearly always fissured also.

hen the brain is developed in excess, it becomes more or less com- ely double. It is very rarely found that any one part is double while remainder of the brain is single; though such is sometimes the case, the gray commissure, for instance. The cerebrum and cerebellum etimes have an unusual number of lobes, and thus appear to be deve- d in excess.

2. *Deviations of Form.*—The form of the brain generally corre- ds to that of the skull, but anomalies occur, principally in the cere- n. In the first place, the brain is subject to variations, in respect to ength, its breadth, and its height: in the next place, some of the er portions of it, the anterior or posterior lobes, for instance, may ed, or come short of, their normal development: the two halves of brain may be unsymmetrical, in consequence either of inequality of generally, or of a difference in a particular diameter, or in conse- ce of a change of relative position in the horizontal or in the vertical ction, &c.; and lastly, its form varies in respect to the stronger or ter marking out of the separate lobes, and to the number, depth, symmetry of the convolutions of the cerebrum, and laminæ of the bellum.

lterations of shape, similar to these congenital deformities, occur , as results of disease of the brain: the principal instances are those hich the symmetry of the two halves is deranged, by an increase or inution in the volume of the whole, or of part of one side, as well as lattening, and loss of the convolutions, &c.

he most striking deformity of the brain is that in which the cerebrum ngle; it occurs in conjunction with cyclopia, and with partial or total nce of the face. (Ateloprosopia; Aprosopia.) The brain forms a le sac, open behind, but completed by the arachnoid, and filled with m: its posterior lobes are so defective, that the cerebellum, corpora rigemina, and even the optic thalami, appear uncovered; whilst y other parts, the fornix, corpus callosum, septum, small commis- s, &c., are also wanting. The cerebrum is sometimes thus single in s of hemicephalus, and this combination associates the latter with opia. (Otto.) Another anomaly, observed in the interior of the n, is allied to that which has just been mentioned: it is that in which optic thalami and corpora striata of the two sides are fused together he middle: a double or a large commissura mollis is a modification he same anomaly.

rom the opposite condition, deficiency of the commissures, a division he brain results. The fornix is very rarely wanting, while, on the other hand, deficiency of the soft commissure of the optic thalami is not unfrequent.

The other smaller organs of the brain are rarely misshapen from any fault in their original formation.

§ 3. *Anomalies of Position.*—Disregarding, for the time, the displacements within the skull to which the brain is subjected by various growths, we find the most striking anomaly in respect to the position of the brain to be hernia,—encephalocele,—extrusion through an aperture in the skull.

Congenital hernia of the brain is occasioned by an extreme increase of the organ in bulk. In most cases it is undoubtedly the consequence of hydrocephalus (hydrencephalocele), the pressure of which interferes with the development of the bones generally, and, at some particular spot, arrests it altogether. The size and form of the congenital hernia of the brain bears a certain relation to the dimensions of the aperture in the skull, as well as to the quantity of the protruded cerebral mass, and of water accumulated in it: the size of the hernia, however, and the dimensions of the aperture in the skull, are very often proportioned inversely to one another. The hernia is sometimes as large as the head, or larger; more frequently it is below that size. Its form is that of a round tumor, or of an appendage to the skull; and when it is large, and the aperture in the skull small, it appears attached by a neck or pedicle.

The protruded mass of brain is covered externally by the general integuments, which are mostly thin, and without hair; internally, the inner cerebral membranes are in immediate contact with it, while, between the two, the pericranium and dura mater are intimately united with each other. True hernia of the brain must not be confounded with saccular protrusions through the skull, which, though similar, are merely herniæ of the arachnoid: they are sometimes so far combined with hernia of the brain, that, as they increase, a portion of that organ may project within their pedicle.

The protruded portion of brain is sometimes in the same state of destruction, and consecutive malformation or vitiation of its growth (Verbildung), as in the case of hemicephalus, the hernia is then combined with hemicephalus, the former passing into the latter.

The situation in which hernia occurs is also very various. Most commonly it is at the occiput; and next, though much less frequently, it occurs further upwards in the mesial line, at the anterior fontanelle. More rarely still, it happens in the lateral regions of the skull, and on the forehead: while sometimes, but most unfrequently, the brain protrudes into the nostrils, or sphenoidal sinuses, and forms a tumor at the root of the nose, or in the pharynx.

The skull, in these cases, is altered in size and shape. As more of the brain protrudes, the cranium becomes generally smaller, and its vault flatter; and if, at the same time, the aperture be large, that form of the head predominates which is exhibited in hemicephalus. But this rule has its exceptions; for if the hydrocephalus be very large, the great quantity of the serum may, in spite of the size of the tumor, not only preserve the skull at its normal dimensions, but even enlarge it beyond them. When the hernia protrudes through the cribriform plate into the nostrils, the vault of the skull sinks, in the form of a saddle.

Hernia of the brain rarely comes on after birth, for it is then only through accidental openings of the skull and dura mater, or those made designedly by art, that the brain can protrude. The hernia is effected by the congestion and turgescence of the brain, which are excited by the external injury, by swelling of the brain arising from acute œdema, by acute hydrocephalus, &c. The protruded portion of brain takes the form of a sausage; it sometimes reaches a considerable size, and measures several inches in length. It is liable to be strangulated by the aperture in the skull and dura mater, and then frequently becomes congested, and mortifies; and injuries to its free extremity may be followed by inflammation and suppuration.

§ 4. *Deviations in Size.*—Many individuals present peculiarities in respect to the size of their brain; but the organ is subject to other and more essential deviations from its natural bulk. I shall treat first of unnatural excess, and then of unnatural diminution of its volume.

1. *Unnaturally large size of the brain.*—Many morbid conditions augment the volume of the brain, as hyperæmia, hydrocephalus, and œdema, adventitious growths, and hypertrophy, or a combination of any of them with one another. I have here to treat of increase of volume by hypertrophy, which is the most important of these morbid conditions; the others have already been, or will be, mentioned in their place.

Hypertrophy of the brain.—Its general characters are, unnatural size and weight of the organ. It varies in degree; and its importance depends partly upon this variety, but mainly on the condition of the skull. The most serious conditions under which it occurs, are when the hypertrophy is far advanced, and the sutures are closed, as the skull then resists the increase of the volume of the brain.

The best plan will be to begin by depicting such a case.

When the skull-cap is removed, the brain, closely covered by the dura mater, swells up palpably (turgescirt); on slitting open the dura mater, the swelling is still more distinct, and it costs some trouble to fit the skull-cap on again. All the membranes of the brain are remarkably thin; the dura mater especially is delicate, pale, reddish, and transparent. The inner membranes lie close upon both the dura mater and the surface of the brain. Their lack of the fluid which usually moistens the arachnoid, and occupies the tissue of the pia mater, is quite conspicuous; they are dry, and their vessels are bloodless and flattened.

Before further dissection and comparison the cerebral hemispheres appear large. Their convolutions are compressed and flattened, and the sulci between them are scarcely discernible.

The usual horizontal section through the hemispheres, a little above the level of the corpus callosum, displays a centrum ovale of unusual size.

The ventricles are remarkably small.

When the whole brain is removed from the cranium, the size of the cerebrum again arrests attention, especially when compared with the cerebellum and other parts at the base of the brain, and with the nerves.

It is quite clear throughout the examination that it is the white substance that is increased in volume, the white substance of the cerebral hemispheres. The cineritious matter is generally of a pale grayish-red color, the medullary is always dazzling white, and remarkably pale and anæmic; a circumstance both of interest and importance, because it distinguishes the increase in the volume of the brain occasioned by hypertrophy, from that which is produced by congestion.

The consistence of the hypertrophied white substance is quite peculiar; it is elastic, and has the somewhat firm resisting feeling of rising dough.

Having thus sketched the most essential and most striking of the appearances in hypertrophy of the brain, I proceed to detail some results which attend it in advanced degrees of the disease, and at particular periods of life.

a. When the hypertrophy is very far advanced, and the sutures are closed, the pressure sets up some absorption at the inner table of the skull, and it becomes rough: the absorption may even go so far as to make the wall of the cranium distinctly thinner than natural. Inferiorly the cerebellum and the structures at the base of the brain are flattened and spread out, evidently by pressure from above. The absorption of the inner table generally begins and goes farthest at the vault of the skull, though it appears indeed, to be most advanced at the base, as the bone, which was there originally thin, is in some parts perforated with holes, produced by the absorption; the orbital and cribriform plates, and the roof of the sphenoidal sinuses, are thus perforated.

β. Enlargement of the skull, as a consequence of hypertrophy, takes place only in the child; but it occurs, whether the bones be held together by interstitial membranes still, or by sutures. The enlargement of the skull, and the hypertrophy of the brain, vary together. In its general form, the skull resembles the hydrocephalic skull.

γ. In some rare cases, among children in whom the disease has rapidly advanced to a considerable extent, the sutures of the skull become loose and separate, especially at the upper part of the head, and the sutural cartilages become suffused, and are of a reddish color.

Hypertrophy of the brain is sometimes congenital, and is then often combined with hydrocephalus; it more usually comes on during extra-uterine life, but is almost exclusively confined to the period of childhood. It is occasionally met with about the time of puberty, and sometimes even in manhood; but at the latter period it is extremely rare.

Congenital hypertrophy is accompanied by very various degrees of arrested development of the vault of the skull, and sometimes, indeed, that part is entirely wanting (acrania), it is attended, also, by general dwarfish growth, and by various faults in the development of the brain, as well as of other organs. When it comes on in childhood and at puberty, it is combined with general enlargement of the lymphatic glands, and but partial obliteration (involution) of the thymus gland: in childhood it is also combined with rickets and a feeble muscular development. And it supervenes upon hydrocephalus, both the congenital and the acquired.

Hypertrophy of the brain usually destroys life with symptoms of pressure on the organ; especially when that condition exists on which I have laid so much stress, viz., closure of the skull. Its course is generally chronic, but not unfrequently it is somewhat acute: the cause of e acute symptoms is found in the fact, that the process, having for me time progressively increased in intensity, at last occasions a rapid d tumultuous addition to the bulk of the brain; but the acuteness is ly apparent, for the disease passes unobserved through its first stages, d at length gives rise to severe and rapidly accumulating symptoms, hen it has gone on to a considerable extent.

Children will tolerate this disease, even though it advance to a consi-rable degree; and after having occasioned a corresponding enlarge-ent of the skull, it may continue throughout life unaltered. This, no ubt, is the explanation of those cases of unusually heavy brain and rge-sized skull, which are occasionally met with in adults: they are ses in which the size and hydrocephalic form of the skull have given se to the idea that a foregone hydrocephalus has been cured by hyper-ophy of the brain. (Comp. 273.)

Hypertrophy is usually a primary and idiopathic disease; frequently, wever, it is secondary, and occasioned by some other disease, more pecially by morbid growths; and of these tubercle and cancer are its ost frequent cause. Its existence, as a secondary disease, has been therto almost unnoticed. But the increase in the volume of the brain, these cases, is so clearly stamped with all the characters of hyper-ophy which I have delineated above, and the frequently inconsiderable lk of the morbid growth so entirely precludes our accounting for these aracters by any compression it could exercise, that there can be no ubt such a form of hypertrophy does occur. The brain is subject to swelling, which, no doubt, belongs to this class, in consequence of the essure exerted on the medulla oblongata when the first two cervical rtebræ are carious and dislocated. This form of increase of volume ust, of course, be accurately distinguished from the swelling produced congestion and œdema.

Lastly, the hypertrophy which has been the subject of the foregoing marks, is a disease affecting the cerebrum, and especially its white bstance. It is rather a frequent disease; not so, however, with hyper-ophy of smaller portions of the brain.

Although the latter be an extremely rare occurrence, there can be no estion that smaller portions of the brain really are separately hyper-ophied. Many of the observations brought forward as instances of is occurrence, are undoubtedly erroneous; adventitious formations in-trated through the cerebral tissue, may have occasioned at once the largement and the error. There are, however, some instances which ay be relied on, in which the optic thalamus and the pons were hyper-ophied; and I have myself met with a most remarkable case of hyper-ophy of the medulla oblongata.

With regard to the question, to be decided by theory and microscopic amination, as to the nature of the added material upon which the crease of volume depends, I have formed the following opinion from peated investigations:

(1.) The disease is genuine hypertrophy.

(2.) It consists as such, not in an increase in the number of nerve-bes in the brain from new ones being formed, nor in an increase in e dimensions of those which already exist, either as thickening of their sheaths or as augmentation of their contents, by either of which the nerve-tubes would become more bulky;—but,

(3.) It is an excessive accumulation of the intervening and connecting nucleated substance.

The immediate cause of hypertrophy of the brain may certainly be congestion; but this is by no means sufficient to explain its occurrence. Regarding it from a higher point of view, I am led rather to believe that the hypertrophy of the brain, and the diseases combined with it, especially the excessive development of the lymphatic system, constitute one disease which is based upon some peculiar state of constitution and mode of growth prevailing chiefly in childhood; a belief which is entertained by others also, especially by Münchmeyer.

2. *Unnaturally small size of the brain.*—Such a condition of the brain may, in the first place, be a consequence of some fault in its original development. The whole brain is then small, but the cerebrum is evidently the most diminished, and its convolutions may not be discernible (microcephalus,—congenital idiocy). Or it is some smaller portion, one of the pairs of cerebral organs, for instance, the development of which, in mass and volume, has been arrested: the anterior or posterior lobes of the cerebrum, on one side or both, the whole cerebellum, or one of its hemispheres, &c., may be thus diminutive.

But far more frequently, the small size of the brain is acquired at a different period of life. It consists then of an arrest of the development of the brain in bulk, in consequence either of premature closure of the sutures of the skull, or of the pressure of the fluid in meningeal hydrocephalus, or of various similar local impediments from without, or else of others, such as dropsy of the ventricles, which operate from within. Sometimes it consists of atrophy of a brain already fully developed. I proceed to a special notice of the last.

Atrophy of the brain.—I may remark, in passing, that the same causes which have been mentioned as interfering with the development of the brain, such as chronic hydrocephalus, or local pressure, may occasionally produce atrophy, either of the whole or of part of it, and proceed to the consideration of the more important instances of atrophy, those which are primary and idiopathic, and those which are consecutive and secondary.

Idiopathic primary atrophy is an affection almost peculiar to old age, and is, within certain limits, a natural process of shrinking or decay (involution) of the organ (Atrophia cerebri senilis,—Senium cerebri). It becomes, however, a pathological condition, even in old age, if it proceed to a very great extent; and still more, if it come on prematurely at an earlier period of life. (Senium—marasmus cerebri præcox.)

This atrophy is confined to the cerebrum. Cazauvielh asserts that the cerebellum retains the full size in the aged, which it had reached in the younger person, at the completion of growth. In contrast, however, with the local, or partial atrophy,—that of separate parts of the brain,—it may be regarded as a total atrophy, or atrophy of the whole brain.

The cerebrum is diminished in volume and weight. The former is ascertained from the existence of a new space between the surface of the cerebrum and the skull, and from the enlargement of the ventricles.

The convolutions are thinner than natural, and the sulci between them broader. The gray substance is of a dirty, or rusty-brown color, running into yeast-yellow. Its consistence may be normal, or distinctly softer than natural. The fibrous substance has lost its pure whiteness, and has a dirty white tinge; it is denser, too, than natural, and is sometimes as tough as leather. This increase of density (sclerosis) is most marked near the ventricles. The ventricles are dilated, their lining membrane is generally palpably thickened, and is frequently covered with a very fine granular, gritty layer, of a crystalline transparency, or an opaque whiteness (p. 272).

More advanced degrees of atrophy and concomitant induration present the following appearance: The surface of a section of the hemispheres shrinks and becomes concave; and, here and there, certain portions offer more resistance than others, and wrinkle and lie in folds: sometimes these firmer portions surround the orifices of the vessels divided in the various sections, and the tissue around the end of the vessel shrinks, and encircles it with a little hard puckered projection.

The fibrous arrangement of the pons, crura, and similar white structures, is rendered more distinct than usual by superficial grooves running in the direction of the fibres.

The porous condition of the white structure of the hemispheres, and of the corpora striata, called by Durand-Fardel "état criblé," is also very well marked. It is equally the result of the atrophy of the brain, and of the congestion of the cerebral vessels to which the atrophy gives rise; it consists of an enlargement of the canals in which those vessels traverse the brain.

The vacuum within the skull, produced by the shrinking of the brain, is filled up chiefly by a clear colorless serum, which accumulates in the sac of the arachnoid around the brain, in the tissue of the pia mater, and in the ventricles. For the same reason, the vessels of the pia mater become varicose, the membranes of the brain increase in volume, and bone is even deposited on the inner table of the skull, especially around the anterior cerebral lobes within the frontal bone.

This form of atrophy, as I have already remarked, occurs in old age; but it may also come on prematurely, and be associated, or not, with manifest senility of the entire organism. When thus premature, it sometimes arises spontaneously; but it is more frequently met with as a consequence of mental diseases which are characterized by excitement, and as the physical cause of the imbecility which follows them. It is one of the results of repeated intoxication and attacks of delirium tremens; and it ensues upon repeated attacks of apoplexy, especially peripheral apoplexy, and upon the processes by which they are cured. Inflammation, too, will bring it on, especially that which is peripheral and its termination in induration, &c.

It is in itself a very important condition, but it becomes still more so from its immediate and further consequences. Those consequences are as follow:

a. Congestion of the brain.—Hyperæmia ex vacuo. These congestions give rise to the transient or protracted attacks, which simulate apoplexy, and are so frequent in old age.

β. *Actual apoplexy*, hemorrhage, finds one of its chief causes in atrophy of the brain, and the congestion to which atrophy gives rise.

γ. *Œdema of the brain* is a very common appearance in the atrophied brain of the aged and imbecile (Calmeil); it may be chronic, or, to a certain extent, acute; and it is attended by effusions into the ventricles.

Atrophy, when it involves the whole brain, and has reached a certain degree, terminates fatally, either by paralyzing the brain, or through some of the consequences described above.

Partial atrophy is very rarely a primary and idiopathic disease; much more commonly it is secondary; it presents two orders of cases:

(1.) Those of the first order are represented by two pretty frequent examples; on the one hand, by shrinking of the optic thalamus and corpora quadrigemina, in consequence of blindness; and, on the other hand, by that of various sets of white fibres after attacks of apoplexy and inflammation, especially when these attacks have taken place at the surface of the brain. The atrophy of one or more parts of the brain, therefore, is consecutive, and arises from disease of the central or of the peripheral extremities of the nerves. The diseases of the peripheral nervous system, which produce such a result, are primary and secondary paralyses, the original physical cause of which, though various, and for the most part unknown, may, in the end be reduced to wasting of the texture of the nerves. The atrophy of the optic thalamus, &c., in consequence of paralysis of the retina, makes it probable, that many of the atrophies of particular parts of the brain are not the cause of peripheral paralysis, but rather the consequence of it; *i. e.*, that the atrophy is propagated from the periphery to the corresponding centre in the brain. The principal diseases of the central extremities of nerves are wasting of the cerebral substance after inflammation and apoplexy, of which I shall have to speak hereafter.

(2.) To the second order of cases belong those losses of substance, those atrophies, which result from inflammation and apoplexy; in which so much of the substance of the brain as was broken down by extravasated blood, or disorganized by inflammatory processes, is altogether removed by absorption, together with the extravasation and the exudation. These are instances of secondary atrophy, occasioned by previous disease of texture. And they are cases, which, especially when the loss of substance is at the periphery of the brain, give rise to the atrophy of different sets of the fibres, as I mentioned among the first order of cases: they may even be so extensive as to occasion wasting of an entire hemisphere, and of the fibres continuous with it in the crus, the pons, the medulla oblongata, and the medulla spinalis.

As these cases of atrophy proceed, they always give rise to well-marked induration (Sclerosis) of the portion of brain which they involve: and that half of the brain in which they occur, and even sometimes the whole brain takes part in the induration too. Atrophy of an entire hemisphere of the cerebrum produced in this manner, is sometimes concentric; sometimes its lateral ventricle is dilated, and then it is excentric. The former, or the case in which the hemisphere shrinks upon itself, is the more usual of the two. In the latter there is a one-sided dropsy of the ventricles.

When the atrophied portion is seated on the surface of the brain, the vacuum is filled up by thickening of the adjoining part of the membranes, by œdema and bulging of the pia mater, by enlargement of the collateral ventricle, and even by the wall of the skull sinking in. When it is deeply seated, its place is, under certain circumstances, after apoplexy, for instance, occupied by serum; and the cavity thus formed exists for some time, and may even continue permanently.

Cases of partial atrophy sometimes bear a very close resemblance to those in which the brain being originally of small size, or defective in some of its parts, consequently presents an originally unsymmetrical appearance. But the atrophy may be distinguished by the induration which it produces in the part which is diminished in size, as well as by the induration of the cerebral substance around, while the vacuum and the complementary serous effusions distinguish it when the skull is found symmetrical, and the rusty-brown, or yeast-yellow coloring of the brain determine when apoplexy has preceded the atrophy, &c.

§ 5. *Solutions of Continuity.*—Amongst these are classed in the first place, various incised, punctured, and shot wounds, contusions, and lacerations of the brain: the latter are produced partly by the instrument with which the injury is inflicted, and partly by the splinters of bone which are depressed or driven in; and partly they are independent of both these causes, and are the result of the violent concussion which the skull has sustained. They are found most commonly at the seat of the injury, but sometimes at other distant points also, deep in the brain, or as is more frequently the case, just opposite the injured spot; and sometimes they exist at several of these points together. The condition of the brain at these parts, varies according to the amount of the injury: either there is no perceptible solution of continuity, but the brain is more or less suffused, that is, sprinkled with red dots and streaks of extravasated blood (capillary apoplexy), and uniformly tinged also of a deep red color with blood; or it is distinctly ruptured, and the intervals are filled with extravasated blood; at other times, the cerebral substance is bruised to a uniform pulp, and of a paler or a darker red color, according to the quantity of blood extravasated into it.

Moreover in sanguineous apoplexy, the brain undergoes spontaneously a laceration, a breaking down, a concussion, exactly similar to that produced by mechanical injury. In the higher degrees of dropsy of the ventricles, as I have already mentioned, the septum may be perforated, the soft commissure torn, &c.; and in some rare cases (see p. 276) the cerebral substance around the ventricles bursts, and the serum escapes.

Incised and punctured wounds, when slight and clean, sometimes heal by adhesion; and the slighter cases of contusion are repaired in the same manner as hemorrhage in the brain; but more extensive injuries give rise to greater degrees of inflammation, to suppuration, and yellow softening, and sooner or later destroy life. Wounds from which loss of substance ensues, heal by granulation, which fills up the cavity, and adheres to the cicatrix that supplies the place of the cerebral membranes and bone. And in this case, the space within the skull is also partly filled up by an enlargement of the ventricle of the same side, proportioned to the quantity of substance lost, and to the difficulty of replacing it with granulation and cicatrix. Sometimes the granulation is exuberant, and grows up to, and beyond the opening in the skull, presenting a condition long known to surgeons by the names of fungus cerebri, and hernia cerebri.

There is one case which calls for especial remark in this place, namely that of concussion, without any perceptible lesion of the continuity of the brain. It results from a fall from a height, and is usually combined with extensive shattering of the skull. It is rapidly fatal; and after death the brain is frequently, though not always, found manifestly collapsed and bloodless. But it may also be produced by force applied to a circumscribed portion of the skull. A solution of continuity, often inconsiderable, then ensues in the skull, a small extravasation occurs over or under the dura mater, and subsequently inflammation, necrosis of the bone, and inflammation and suppuration of the dura mater take place at the injured spot. These are not unfrequently preliminaries of a diffused inflammation of the pia mater (meningitis); though sometimes the meningitis comes on without such antecedent. But the gravest of all are those rarer cases in which, not only without any of these occurrences in the neighborhood of the brain, or perceptible lesion of its continuity, but even, without the slightest subsequent alteration of its structure that can be detected, an affection of the brain supervenes, either immediately upon the injury or some time after, and proves fatal. In some cases of this kind, which are well known to distinguished surgeons, as well as in others in which the violence inflicted was attended with palpable structural disease of the brain, I have observed not only a remarkable emaciation of the whole body, but more especially a very rapid disappearance of the mass of the blood (anæmia), which was the more conspicuous from the previously full habit of the patient.

§ 6. *Diseases of Texture.*

1. *Hyperæmia,—Anæmia.*—Congestion of the brain is a very common appearance; and it is generally associated with a corresponding degree of congestion of the pia mater.

Its anatomical characters are—first, injection of the cerebral vessels, and the appearance of an unusual number of bloody points on the cut surface of the brain. The gray substance, when but slightly congested, exhibits some shades of red, and in young persons, and especially in the child, it presents distinctly a bright-red color. In intense congestions, the fibrous substance loses its clear whiteness, and, in children particularly, acquires a grayish-red, and in some very rare cases a red hue. The brain is swollen; but its increase in size is distinguished from that enlargement which occurs in hypertrophy of the brain (p. 288), by the turgid condition which has evidently given rise to it. Bouillaud has called attention also to a firmer consistence of the brain, as occurring in congestion; it is, however, by no means a constant, and still less an essential appearance.

Hyperæmia of the brain takes place in the course, or follows as a consequence, of very various acute and chronic diseases. It is sometimes active, sometimes passive; and it may be mechanical. Occasion-y it arises from none of these causes, but comes on independently; d then it may be transitory or persistent, or may recur and become bitual. Congestions of the last kind occur especially in childhood d at the period of puberty. Hyperæmia is an important condition ien it is produced and kept up by structural disease, as may readily be served in all new formations in the brain and cavity of the cranium, d especially in highly vascular turgid structures, like cancer of the ain. Those congestions, also, which result from the existence of a cuum in cases of atrophy of the brain, and which were referred to at ge 287, are of considerable importance.

Among the consequences of hyperæmia, slight and repeated conges- ns may be mentioned as leading, especially in childhood, to hyper- phy of the brain. Another frequent and a fatal consequence of it is lema of the brain, which may be acute or chronic, according to cir- mstances, and attended with an effusion of serum into the ventricles. oreover, there is no question that hyperæmia proves fatal in cases own as vascular apoplexy. Such a result is met with in the course of ny acute diseases, which give rise to local congestions. Diseases of e brain itself, especially hypertrophy, and different new formations thin the cranium, the pressure of which displaces the organ, often oduce sudden death by the congestion to which they give rise; and, stly, diseases of the lungs, the heart, and the great vessels, which struct the circulation, and especially those which prevent the free rrent of venous blood towards the heart, are frequently terminated by chanical congestions.

It is a question of much importance, whether the frequent cases of dden and unexpected death in previously healthy persons, in which the ly or the principal post-mortem appearance is a certain amount of ngestion, are produced by this congestion, and are to be considered as ses of palsy of the brain from hyperæmia, whether in fact such con- stions are sufficient to cause death or not.

a. In answer to this question, I may remark, that, in a certain number the cases referred to, this congestion is the only morbid appearance the body, and has reached a degree which, in the present state of our owledge, justifies the conclusion that the brain has been paralyzed by But the number of such cases is comparatively very trifling.

b. In a considerable number of cases, again, moderate congestion of e brain is found associated with hyperæmia of the lungs. It is scarcely ssible to say which of these conditions was the primary, and which the condary, whether they did not spring both together from the same urce, and which of them actually produced death. But as it is quite mmon for congestion of the lungs to be the only morbid appearance cases of sudden death, and as it is decidedly the more marked appear- ce when cerebral hyperæmia is present also; we may, in determining e mode in which death has taken place in these cases, conclude that e congestion of the brain is usually of secondary importance.

c. Besides cases of these two kinds, there is still a number of others which all that is discovered upon examining the body is so slight a ngestion of the brain, that it would not be thought of, if any other orbid appearance presented itself. The mode of death in most cases, and especially of sudden death, is still too little understood to allow us to say positively whether these congestions are the efficient cause of it, or are merely accidental phenomena, and dependent on the agony: but perhaps we may say that there is an individual tendency of the brain to palsy (Lähmungsfähigkeit), just as there is an individual liability to death (Sterbensfähigkeit), and so incline rather to the former opinion that in certain persons such congestions may prove fatal.

Persons of what is called an apoplectic habit, are far less subject to congestions of the brain, and particularly to vascular apoplexy, than those of an opposite conformation, and than children.

Anœmia of the brain is a highly important condition, and one very dangerous to life. It is usually a local part of the general bloodlessness produced by hemorrhage, or by the consumption of the blood which takes place in the course of acute and chronic diseases. A very remarkable instance of anæmia is that which arises from the contraction or obliteration of the vessels, which convey blood to the brain: it is affected by deposition on their inner walls, and may occur at any part of their trunks, or at their orifices in the arch of the aorta. (See Anomalies in the Calibre of Arteries, vol. iv.) It is a very important condition also when it results from hypertrophy, swelling, compression, or displacement of the brain in cases of sanguineous apoplexy, inflammation, yellow softening, or new formations.

2. *Cerebral hemorrhage.*—Hemorrhage (Apoplexia sanguinea; Apoplexia gravis) is a very common disease in the brain, and is often suddenly fatal. I class it with hyperæmia, although it is not necessarily accompanied or caused by striking congestion.

Cerebral hemorrhage consists in an extravasation of blood into the substance of the brain, and a solution of its continuity corresponding in extent to the amount of the bleeding; the latter injury consists generally of breaking-down, laceration, or contusion of the cerebral substance.

The intensity of the hemorrhage, the number of the bleeding vessels, and the nature and extent of the disorganization of cerebral substance occasioned by the extravasation, produce many marked varieties in the form of the seat of hemorrhage.

Sometimes a spot of gray or white cerebral substance, varying in extent, is speckled or striped with a small number of dark red dots and streaks of extravasated blood (ecchymosed): the streaks run parallel to the nervous fibre, the intermediate cerebral substance preserves its normal color and consistence, and, where the fibres of the brain run in one direction, it seems to the naked eye merely drawn asunder, some few only of the elementary parts having suffered an actual solution of continuity.

At other times the number of these extravasations is more considerable: they lie closer together, and some of them having coalesced, are thereby increased in size. The cerebral substance appears to be uniformly suffused, and colored with various shades of red: it is of a soft pulpy consistence; and, apparently, in consequence of the number of original small extravasations, and still more by the confluence of larger ones, it is broken into shreds, and softened to a red pap.

When this is the case, the small extravasations become more numerous, l, still coalescing, produce more and more extensive destruction of the ermediate cerebral substance; and thus, as the other extreme, we find the primary small extravasations united in a single great one, which ludes the broken-down interstitial substance of the brain, and is itself tained in a cavity of lacerated, and bruised cerebral tissue.

This, therefore—the apoplectic cavity (Herd)—proceeds from hemor- ges occurring at several points at once,—from the confluence of many all cavities. The form first described has received the name of Capil- y Apoplexy.

An extensive cavity is, however, sometimes formed in another manner, ., by the independent enlargement of a single small extravasation in sequence of the continuance of the hemorrhage; for there are some gle and very small extravasations, of the size of a poppy- or millet- d, which, when the hemorrhage continues or recurs, increase rapidly by degrees, and tear and separate the surrounding cerebral substance various directions. Though the tissues composing the walls of such a ity, may resemble the broken-down structures already described, t the extravasated blood which it contains is, in its interior strata, at st, free from all admixture of cerebral tissue.

It is not clear whether one form of the hemorrhagic cavity may, from y particular morbid cause, be occasioned by the other; but it appears, ertheless, as if that described second, were principally dependent on pertrophy of the left ventricle, and gave rise to the large (foudroy- tes) cavities which prove suddenly fatal.

The apoplectic cavity, whether produced in one way or the other, sents varieties, when recent, which are important in several respects.

Seat of the Hemorrhage.—It happens in the cerebrum far more fre- ently than in any other portion of the brain. The cerebellum is compara- ely very seldom attacked, and the pons yet more rarely. Its occurrence the corpora quadrigemina, the pons, and the medulla oblongata is ite exceptional, and it almost never happens in the corpus callosum, the nix, and the hippocampi. There is, however, pretty frequent excep- n to this rule: for when a large cavity is formed in the cerebrum, one more small secondary cavities are found also in other parts of the in, especially in the cerebellum and the pons.

In the cerebrum, the situations in which hemorrhage principally occurs the optic thalami, and corpora striata; from them the white substance the hemispheres is invaded: the gray matter of the convolutions also a common site. The cavity is less frequent in those fibrous parts of hemispheres which are distant from the seats of gray matter.

From this it is evident that the masses of gray matter, and those por- ns of the brain which contain considerable quantities of gray matter, eminently the seat of apoplexy. It is unnecessary, after stating se general facts, to prepare a scale of frequency.

The distinction of apoplexy into *peripheral* (that of the cortex of the in), and that occurring in the central parts is worthy of notice, inas- ch as the former is followed by physical changes and functional dis- bances of considerable importance. When spontaneous it almost ays occurs only on the convexity of the hemisphere.

Hemorrhage into the lateral ventricles,—not the rupture into the ventricles, which so frequently happens to apoplectic cavities in the corpus striatum and optic thalamus, but hemorrhage from the lining membrane, and the vessels ramifying near it,—is an extremely rare occurrence. I once met with it, as others also have done, in a very large congenital hydrocephalus.

The cavity varies in *size* from that of a poppy, or millet-seed, to that of a man's fist, and even beyond, so that at length it may include an entire hemisphere of the cerebrum. This part is of course the seat of the largest cavities. It not unfrequently happens that they burst through the brain, either outwards on its surface, or more commonly inwards, into the cavity of the lateral ventricle, as might, indeed, be expected from their being more usually situated in the optic thalami and corpora striata. The consequence in the former case is extravasation, first, into the tissue of the pia mater, and then between it and the cerebral layer of the arachnoid, to a greater or less extent. Not unfrequently the whole of the pia mater surrounding the diseased hemisphere is suffused, and free extravasation is poured out even into the ventricles. In the latter case, the first and immediate result is the effusion of a large quantity of blood into the collateral ventricle; it passes into, and fills, the opposite ventricle, producing at the same time, much laceration of the septum and fornix. The effusion proceeds into the third, and thence into the fourth ventricles, and from the ventricles into the pia mater all round the brain, but especially at the base. The size of the rent varies; it may be an inconsiderable fissure, or a large cleft, gaping into the ventricle; the latter is especially the case in the corpora striata and optic thalami.

In peripheral apoplexy, or apoplexy of the superficial part of the brain, the cavity is covered in, and closed by the pia mater, and the tissue of that membrane is suffused. In superficial extent, peripheral apoplexy usually includes the space of about a square inch.

The smallest cavities occur in those parts of the brain, in which apoplexy is, in general, less frequent, although some cavities, of the size of walnuts, are not very rare in the pons, and even larger still in the cerebellum.

The *number* of recent cavities is also various. Usually but one is found, though to this there are frequent exceptions. With very large and suddenly fatal cavities in the cerebrum, one or several others, in different parts of the brain, are very commonly associated, which are secondary to the former in point of size. It is also interesting to observe, sometimes, in the very atrophied brain of old persons the simultaneous, or nearly simultaneous, occurrence of very numerous small cavities: they equal millet-seed, hemp-seed, and even beans in size, and occur in different parts of the brain.

It is also remarkable that two symmetrical cavities are sometimes found in corresponding organs of the brain, for instance in the corpora striata.

The *form* of the cavity, when small, is generally round or elliptical; in fibrous parts, like the pons, it is very often a slit parallel to the fibres: larger cavities are more irregular, and are dilated into pouches in various directions.

e parts around, constituting the *walls* of the cavity, consist of cere- substance more or less extensively suffused, red, and broken down ı soft and very moist pulp: in large cavities, ragged shreds of it into the cavity.

e *contents* of the cavity, too, the extravasated blood, present numer- arieties. In the first place, as has been already explained, it may, ıy not, enclose shreds of the destroyed cerebral tissue. Its quantity sponds with the size of the cavity, and may amount to six, eight, or ten ounces.

ere are many diversities both in the *degree and the manner of its ılation*. Sometimes the whole extravasation becomes one uniform , blackish-red mass; at other times it is partly fluid, and partly in of various consistence: occasionally there is a more marked separa- nto a fluid portion and one coherent cake; or the entire mass has ıaled into a sort of placenta that fills out the cavity and assumes its Here and there, moreover, the fibrin may be separated in the of clots, or cords, or membranes, which pervade the blackish-red It is of much importance to remark those forms of coagulation, itherto observed, in which the fibrin, when in considerable quantity, ›osited around, and encloses the clot and serous portion—the *peri- ıl* form,—or is lodged as a *central* clot, in the interior of the coagu- mass. Each form opposes permanent obstacles to the healing, that e contraction and closure of the apoplectic cavity.

stly, a brain, which is the seat of apoplexy, suffers not only a solu- ›f continuity at the part itself, but also some displacement corre- ling to the size of the cavity. The cerebral substance surrounding avity is stretched and torn, the segment of the brain containing it arged, swollen, and more or less altered in form, and a portion of swollen by a large cell is found to fluctuate, when much of the d blood has not coagulated. The pons is thus enlarged around a n its centre, especially in breadth; but the changes are most strik- hen one of the cerebral hemispheres is occupied by a large apo- c cell. It is forced against the dura mater and cranium, and inwards st the opposite hemisphere; it becomes prominent and convex to- ı the falx cerebri; it feels quaggy (schwappend,—wabbling); and enclose a very large cavity, it ruptures on the removal of the dura . The inner membranes are thin; the serum which had been pre- ly infiltrated through them, is removed; their vessels are compressed; onvolutions are driven close together, flattened and diminished in the structures at the base of the brain are flattened, the opposite icle is narrowed, and its contents are displaced. When a cavity of ind opens into the ventricle, the opposite hemisphere also shares in nlargement, &c., as much as is possible.

cent apoplexy is followed by numerous changes in the cavity itself, ı cerebral substance around it, and even in the whole brain; and the iption of the recent cell may now be followed by some account of They include, in a word, the terminations and consequences of exy, provided a fatal result has not ensued suddenly, or after some period, such as a few days. I will speak first of the changes which d a favorable result, and constitute the reparative process in apo- . A representation of this process can be obtained only by numerous observations on persons who have died at various periods after an attack of apoplexy. The parts in which these changes occur, are *the extravasated blood*, and the *surrounding cerebral substance*, or wall of the cell.

Numerous changes of color gradually take place in the extravasation; it becomes blackish-red, then brown, of the color of plum-sauce, rusty brown, and yellow, like yeast: at last this color also fades completely, or nearly so; and there remains only a clear and colorless, or a turbid white fluid. Corresponding alterations take place also in the consistence of the extravasation, and in its general composition. They are manifested on the whole in the extravasation becoming fluid, the fibrin and blood-disks being absorbed, and in the progressive changes in the pigment which have been mentioned.

The shreds of cerebral substance contained in the extravasation are also absorbed, and disappear entirely.

Whilst these changes are going on in the contents of the cell, we observe others also in its *walls*.

In the first place that portion of the wall of cerebral tissue which is suffused, torn, and disorganized, and which hangs into the cavity in the form of pulpy shreds, becomes partly absorbed, and partly fluid, so that the inner surface of the wall acquires a polished appearance, and the cavity itself a more regular rounded form. A reactionary inflammatory process then commences in the neighboring uninjured cerebral substance: it is moderate in degree, and its products partly undergo a change of structure, and partly both they, and the cerebral tissue, which is the seat of the process, become reduced to a fine molecular mass, and are gradually absorbed. In its general appearance, therefore, this layer seems to consist of fibrils like cellular tissue, of numerous nucleated structures, some rounded and others elongated into fibrils, of delicate nucleated fibres, of elementary neucleoli, partly separate, and partly conglomerate, and forming the so-called exudation-corpuscles, and of a certain quantity of yellow, or yellowish-red, amorphous pigment. And, according to the amount of one, or the other, the density of the layer varies: if the formation of new tissue predominate, its density is considerable, while in many cases we find it to be made up of honeycombed compartments, the partition-walls consisting of these fibrillated structures, and the intervals being filled up by a thick, white fluid, like lime-water, which is composed of the molecular matter already spoken of. After the absorption of the latter, the layer always increases in density, and appears like a cicatrix surrounding the apoplectic cavity.

Meanwhile the extravasation within the cavity has undergone the changes already generally described. The fluid contains a quantity, varying according to circumstances, of separate or confluent elementary corpuscles, and is dotted with more or less, brown, yellowish-red, or yellow pigment, which may be either amorphous, or in the form of very small prismatic crystals. Moreover the cell is either traversed by a delicate network of varying firmness, which contains this fluid in its intervening spaces, or it forms a simple cavity with its walls lined by a similar network. Minute examination proves this to be a gelatinous blastema, which is gradually resolved into exceedingly fine fibrils, and contains many of the elementary granules, separate or conglomerate, and a greater or less quantity of pigment. It constitutes what is looked upon as the characteristic lining of the cavity when changed to the so-called *Apoplectic cyst.*

In this lining membrane and the equivalent network, just as in the fluid that fills its cells, the pigment sometimes gradually disappears, at least from the naked eye; an exceedingly small quantity remaining perceptible with the aid of the microscope. Indeed, the presence of pigment has less weight in the diagnosis than is ascribed to it, as similar pigments remain after many other processes, which are attended with extravasation, though it be only in the form of the smallest ecchymoses. I may instance, especially, inflammation.

This lining membrane is found at later periods gradually to change into a finely fibrillated cellular tissue: it becomes more compact and smooth on its inner surface, the lining of the apoplectic cyst looking, in fact, like a serous membrane. Sometimes new vessels are formed in it.

The apoplectic cavity is, in general capable of a still further reparative change, or decay (involution): viz., *gradual contraction and finally closure—wasting of the apoplectic cyst.*

This result ensues when the lining of the cyst is sufficiently penetrable to admit of the absorption of its fluid contents. The cyst becomes gradually smaller, especially in the diameter corresponding to the course of the fibres in its neighborhood: and its walls approach one another, and finally unite. The spot where the cyst has existed, is then generally marked by an elongated callus (apoplectic cicatrix), containing frequently, though, as I have already said, not constantly, a streak of pigment in its centre.

The period occupied by these different changes, up to the complete healing of the apoplectic cell, cannot be accurately determined. In general, it may perhaps be said, that the apoplectic cyst is formed within two or three months, but nothing is certain with reference to its subsequent changes and closure. These depend mostly on the size of the cyst.

I proceed to mention the most important of the conditions under which the shrinking and closure of the apoplectic cyst are impeded and entirely prevented.

a. Large cysts generally do not cicatrize; their size certainly diminishes more or less; but after this they generally remain permanently: there is, however, an occasional exception in the case of considerable contraction, and almost, and even quite, complete closure of very large cells.

b. Vascularity of the lining membrane of the apoplectic cyst appears to present an obstacle to this result, as it is not unlikely that a secretion of serous fluid may coexist with absorption.

It is, indeed, not improbable, that under such a condition the cyst may even become enlarged, as a consequence of the atrophy of the brain which follows an attack of apoplexy, and the congestion and increased secretion of the vascular lining resulting from the atrophy.

c. An especial and a permanent obstacle to the contraction and closure of the apoplectic cyst, is found in the fibrin being separated, while the extravasation is recent, in the form of a peripheral, or of a central, clot.

In the former case, the extravasation lies enclosed in a capsule of coagulated fibrin, colored red from admixture with the coloring matter of the blood, just as in the extravasations into the sac of the arachnoid. The changes already described go on in its interior; while externally, in the contiguous cerebral substance, the reactionary process advances to the formation of a callous wall. But the compactness of this capsule of fibrin renders absorption of its contents impossible, or, at least, very difficult; and the cavity is scarcely capable of any reduction in size. In the second case, the fibrin of the extravasation is coagulated into a solid, and generally rounded, mass. It is true, that a certain diminution in the size of the mass is then possible, partly by solution in the serous fluid surrounding it, and partly by the shrinking that attends its conversion into a fibrous tissue: the diminution, however, is not considerable, and occurs but very slowly, and the mass of fibrin offers a permanent obstacle to the complete closure of the cavity.

Such a separation of the fibrin could be expected only in an extravasation which is of large size and rapidly formed; and it is, in fact, only in large cavities that I have observed it. In the so-called capillary apoplexy, the clots are formed by repeated small hemorrhages occurring in different points, and gradually coalescing in a single cavity, and in it these unfavorable modes of separation of the fibrin into large coherent masses, are not likely to occur.

d. Apoplexy occurring at the surface of the brain (peripheral apoplexy), especially when extensive, scarcely ever terminates by a complete closure of the cavity.

The healing process, in these forms of apoplexy, is essentially the same as that already explained. Some differences arise simply from the locality: the walls of the cavity are, cerebral substance on one side, and pia mater on the other; and the callous induration also occupies both the pia mater and the cerebral wall. When the cortical substance is destroyed in its whole thickness by the hemorrhage, the white matter of the convolutions and that forming the grooves, partake of the induration. The lining of pigment also adheres both to the membranous and to the cerebral walls of the cavity. It is generally of greater thickness on the latter, and constitutes, as I believe, the "yellow plates of the convolutions" spoken of by Durand-Fardel. This observer attributes the disease in question to chronic softening (inflammation); and in a monograph which he has issued, he has unduly extended the sphere of inflammatory softening of the brain, and seems, moreover, to have altogether misunderstood peripheral hemorrhages of the brain and their results. In cavities of small extent, complete closure, by cohesion of their two walls, is not unfrequent; but larger ones, as has been already remarked, not only continue for a long time, or permanently, in the condition of the apoplectic cyst, but they become manifestly larger, and give rise at the same time to considerable atrophy of the brain. The cause of this unquestionably is, that the vascular structure composing the outer wall of the cavity, viz., the pia mater, becomes congested in consequence of the vacuum existing within the cranium, and serum exudes from its vessels into the cavity. In the situation of such cavities the membranous wall is seen projecting, in the form of pendent, fluctuating bags.

The cerebrum affords the best opportunity for observing the healing rocess just described. Very large cavities close in this part, and even n the cerebellum also, at least so far as to leave merely a small cavity, r cyst, which in itself is not injurious. In many portions of the organ, specially in the pons, where, on the whole, apoplexy is not very rarely bserved, only very small cavities heal completely. It is generally elieved that cavities, the greater diameter of which is parallel to the ourse of the fibres of the brain, and in which, therefore, the mass of erebral matter is more thrust asunder than really destroyed, heal, that s, close, more readily than those which are oppositely situated. The bservation made above (p. 297) may be connected with this, that the ontraction of the apoplectic cyst takes place especially in the diameter which is parallel to the direction of the contiguous fibres.

Although the process of healing may be going on, and a cavity be in favorable state, it may yet prove fatal secondarily at any period. The urther terminations and consequences of apoplexy may, therefore, be ntroduced here.

a. Apoplexy may be fatal at once, primarily, and suddenly (apoplexie oudroyante), or after a short time, some hours, or a few days: and death, n such cases, results from the extensive destruction of the brain, and rom pressure. Of this kind are large central cavities, those of the size f a hen's egg, and even smaller. Still more certainly fatal are larger nes, which burst into the meshes of the pia mater without, or the cavity f the ventricles within. In certain parts of the brain, again, as the ons, medulla oblongata, corpora quadrigemina, a cavity which is not bsolutely of inordinate size, may prove fatal by its suddenly withdrawng the influence of the brain from vital functions.

b. Apoplexy is fatal secondarily after a short interval, when, a rectionary process having been established in the neighborhood of the avity, the inflammation becomes excessive, and is accompanied by ellow softening of the brain around. This is, moreover, occasionally ssociated with a very acute softening of the stomach.

c. Another secondary, and more remotely fatal result may ensue at ny stage of the healing process when already commenced and advanced; ts symptoms are those of paralysis and imbecility, marasmus and tabes, næmia, and so forth. This latter secondary mode of death is partly ccasioned by those diseases of the brain which are developed in conseuence of apoplexy and its healing process. They are as follow.

α. The first effect of apoplexy is a permanent loss of a portion of the erebral mass.

β. A very frequent, if not an invariable, consequence of this, is a nanifest atrophy extending to considerable distances, in the course of hose fibres which are included in the apoplectic spot. Large peripheral poplexies are particularly remarkable in this respect.

γ. This atrophy itself, together with the diminution and closure of the poplectic cyst, gives rise to a corresponding amount of vacuum within he cranium; and the vacuum becomes greater as repeated attacks of poplexy gradually involve the whole mass of the brain in the atrophy.

δ. The atrophy of the brain, if not followed by œdema, is constantly ombined with induration (sclerosis—condensation, and leather-like hining and toughness) of the white substance, and with the discoloration of the cerebral substance already described in the atrophy of the brain of old persons: and it gives rise to premature marasmus of the brain, and early failure of its powers. Doubtless the induration is principally due to the atrophy itself; it increases with the number of the apoplectic cavities; but it may also further arise from the whole brain participating in the inflammation and healing process of repeated attacks of apoplexy. At least, increase of density and adhesions of the superficial parts of the brain to the pia mater, are very commonly produced by an extension of the process of reaction much beyond the seat of peripheral apoplexies.

ε. The vacuum in the skull produces congestions of the brain, and, thereby, repetitions of the apoplectic attack; as well as chronic, and even acute, œdema, if it should be occasionally increased.

ζ. A varicose state of the cerebral vessels sometimes comes on in the neighborhood of the apoplectic cyst and cicatrix.

All these appearances, either singly or together, are more developed in proportion to the size of the apoplectic cavity, and still more in proportion to the number of attacks. But extensive, and frequently repeated, peripheral apoplexies are, as has been already noticed, most remarkable in this respect; and it is in these that the most marked examples of imbecility and weakness of intellect occur.

In the few cases I have observed of primary hemorrhage into the lateral ventricle, the extravasation had always coagulated into a rounded cake, and was either loose or lightly adherent to the wall of the ventricle.

In conclusion, I turn to the pathogeny of hemorrhage in the brain. It is, in my opinion, entirely within the scope of anatomical inquiry.

The source of the hemorrhage is the finer vessels and the capillaries of the brain, one or several of which are ruptured. The rupture, however, of so large a number of vessels as we observe even in a cavity of only moderate size, is far from being entirely primary; by far the greater number are torn secondarily in the common destruction of cerebral tissue, by the increasing effusion.

The first that meets us in the search for the causes of apoplexy is—

1. Manifest external violence, producing traumatic apoplexy; it may act either directly upon the skull, or indirectly through the trunk, and produce concussion of the brain: in the former case, wounds of the soft parts of the head, and injuries of the skull itself, are usually associated with it. Most frequently these forms of apoplexy are rapidly fatal, by the general paralyzing effect of the concussion upon the whole brain. The mode of their origin is evident.

2. A second class of apoplexies are those which result from different pre-existing anomalies, the mode of action of which may be more or less clear: they are named *spontaneous apoplexies.*

The mode in which these spontaneous apoplexies arise remains now to be examined; and, in order to omit none of their causes, all the circumstances under which they occur, may, I think, be so arranged as to afford a useful survey of the most important facts.

Cerebral hemorrhage is not unfrequently observed in the course of convulsions, especially puerperal convulsions and epilepsy. It is generally very extensive and rapidly fatal.

It occurs also sometimes in the neighborhood of tumors, tubercle, and cancer of the brain.

Occasionally, inflammation and obstruction of the venous trunks of the pia mater, or of the longitudinal sinus, are attended with hemorrhage in the contiguous substance of the brain; it occurs also in the neighborhood of inflammation (red softening), as well as in the inflamed spots themselves.

Sometimes it takes place in pregnant females, and persons with distorted spine, when the lungs are morbidly dense.

In some very rare cases in which congenital hydrocephalus is increasing rapidly after birth, blood escapes either into the cerebral substance or into the cavity of the ventricles from the vessels ramifying on their dilated walls.

Sometimes it occurs in the course, or as the consequence of fevers, particularly the typhus and typhoid.

And occasionally during the progress of Bright's disease of the kidneys (albuminuria).

Apoplexy appears in the body under all these circumstances, both in the form of the cavity, and, not unfrequently, in the capillary form.

Hemorrhage very commonly occurs in the brain in persons laboring under simple hypertrophy, or hypertrophy with moderate dilatation of the left ventricle of the heart. The coincidence of apoplexy with this disease of the heart is so constant as to afford ground for stating it as a rule (constitutio apoplectica cordis).

The arteries are very often found diseased in cases of apoplexy, in the form of bony or atheromatous concretions, and of fatty degeneration and brittleness of their middle coat. The arterial trunks, especially within the cranium, are then also thickened, rigid, ossified, and brittle, as well as enlarged (especially in the form of aneurisma cyrsoideum). Hence it may be inferred, that the more minute arteries, and even the capillaries within the brain, are in a similar condition: especially as the former are, in fact, sometimes found ossified, and the brain appears as if filled with stiff wires. This, of course, very often coexists with hypertrophy of the heart.

The brain is very liable to hemorrhage, if it be the seat of atrophy, whether spontaneous or however produced; but especially of that atrophy which is the result of previous apoplexy.

Finally, it is very common in advanced life, when the two last-mentioned causes are so often present together. All these forms of apoplexy appear in the body as more or less extensive cavities, and they are very often rapidly fatal.

Reflecting on these circumstances under which hemorrhage occurs in the brain, we discover the proximate causes to be, in general, *congestions*, *excessive action of the heart*, and *disease of the bloodvessels*. All, when they have attained a certain degree, lead to rupture of the vessels.

The *congestions* are of various kinds: convulsions produce congestion, which may be active or passive, according to circumstances, and rupture the vessels by the violence of the heart's action.

Apoplexy in the vicinity of tumors, or of inflamed and obstructed veins of the pia mater, or sinuses of the dura mater, or when occurring in connection with morbid density of the lungs, and consequent dilatation of the right ventricle of the heart, is the result of mechanical hyperæmia, whether the cause of the hyperæmia be near the apoplectic spot or remote from it.

Apoplexies which occur during, or subsequent to, typhus and typhoid fevers, arise from the congestions (active and passive) which are notoriously so frequent in the brain, as well as in other organs, in the course of those diseases.

A similar remark applies to the apoplexy met with in the course of Bright's disease of the kidney; in the production of which, however, mechanical influences must also be taken into account, especially the defects which are so frequent in the valvular apparatus of the endocardium.

In the apoplexy of congenital hydrocephalus, the congestions which give rise to the rapid increase of the original affection, and the stretching of the cerebral vessels which results from this increase, both predispose, the latter mechanically, to rupture the vessels.

But the hitherto unnoticed congestions, arising from vacuum in atrophy of the brain, are of by far the most importance. These, when alone, and more especially when combined with brittleness of the vessels, are, without doubt, the cause of the frequency of apoplexy in advanced life, and particularly of its recurrence in some cases.

The hypertrophies of the left ventricle of the heart, mentioned above, occasion rupture of the cerebral vessels, by the violent impulse which they give to the blood.

The disease of the vessels alluded to, involves, as is well known, brittleness, and a tendency to easy rupture of their coats. Such a state of the coats of the vessels appears to some extent necessary as a cause of apoplexy, for it is often observed that the deepest congestions, whatever their nature, but especially those intense mechanical ones which give rise to cyanosis, do not produce apoplexy.

And, further, the last two causes, viz., increased impulse of the left ventricle, and disease of the vessels, have such a reciprocal supplementary influence, that the more developed one is, the less amount of the other seems necessary to occasion apoplexy. Thus, so soon as the disease of the vessels has reached a certain extent, hemorrhages very commonly occur in the brain, without increase of the action of the heart, and during a state of quietude both of body and mind. A similar occurrence has been already referred to, in the instance of spontaneous rupture of large arteries. (See Dissecting Aneurism, vol. iv.)

There is no single cause that will account for the frequent repetition of attacks of apoplexy in many individuals, and its simultaneous appearance at several different spots in the brain, but the presence of disease of the vessels. This also partially explains its happening symmetrically in corresponding portions of the brain at the same, or nearly the same, period. (See the remarks on the symmetrical occurrence of Disease of the Arteries, vol. iv.)

The liability to cerebral hemorrhage is of course greater, in proportion to the number of the adduced causes that are present. The congestion arising after atrophy of the brain from vacuum within the cranium, ypertrophy of the heart, and disease of the vessels, very frequently xist together in advanced life.

Among the predisposing causes, all mention of the so-called *habitus poplecticus* might be omitted. And the omission would be justified by e facts; for the notorious "Constitutio Apoplectica" is a mere hypoesis, which is refuted on every occasion. The apoplectic habit predisoses much more to congestion and vascular apoplexy of the lungs, which, deed, as it induces acute œdema of those organs, is very commonly tal, and is, unquestionably, the most frequent of all the modes of suden death.

I have also made no allusion to *yellow softening of the brain*, as a redisposing cause of apoplexy. But, without entering into any further bservations, I may remark that, though it not unfrequently follows, I o not recollect any case in which yellow softening of the brain, as a rimary disease, had given rise to hemorrhage.

It appears from what has been said, that as the occurrence of hemorage in the brain is entirely mechanical, all the predisposing causes are echanical also.

Though quite convinced that this is the fact, I can scarcely avoid enring upon the question as to the existence of some morbid state of conitution, or dyscrasia, as a cause of apoplexy; especially as analyses of e blood of apoplectic persons have recently been published, the results f which seem to point to the blood as the cardinal predisposing cause of poplexy.

It may be reasonably doubted whether any morbid state of the blood ould have the effect of rupturing vessels without some previous alteraion in the structure of their coats: such a result could ensue only from state of morbid expansion of the blood; such for instance, as occurs in yphus and typhoid fevers, in combination with congestion. I certainly, however, agree so far, that a certain anomalous condition of the blood does coexist with those circumstances under which apoplexy is most frequently observed; only, as we shall see, it never is an essential predisposing cause, but is itself conditional upon and subordinate to the mechanical relations. Such are:

1. The abnormal state of the blood which is developed in the course, or as the consequence, of hypertrophies of the heart—a venous crasis—cyanosis.

2. That morbid condition of the blood, which gives rise to the accumulation of an inner membrane upon the vessel by deposition from the arterial blood, and thereby to the so-called ossification of the arteries: and also that morbid condition which, after deposition has taken place within the arteries, may be occasioned by the absorption of the elements constituting the atheroma of the artery, especially the fat. No accurate information has hitherto been obtained as to the characters of these morbid states of the blood.

3. A change in the condition of the blood, which occurs in advanced ife, and is brought on by atrophy of the brain, but which loses its importance in the presence of the coincident mechanical causes.

The results of the analyses instituted by Andral and Gavarret, on the lood taken from persons suffering from the preliminary symptoms of apoplexy, as well as after the attack, prove clearly the existence of a morbid condition of the blood, but I maintain that they lead to no further result than that the blood of the persons spoken of, is thus composed. So long as more numerous and more widely extended analyses are wanting, it may be asked, without reference to the denial that this state of the blood gives a predisposition to hemorrhage in the brain,

1. Is not this just the condition of the blood which is found in individuals with hypertrophy of the heart, or in those who have ossification and atheroma of the arteries, or in aged persons, and individuals with atrophy of the brain?

2. Has not the disease of the brain itself (apoplexy), either while impending, or after its occurrence, already produced a change in the state of the blood in those very persons whose blood was examined? This question may the more fairly be asked, considering on the one side, the important influence exercised by the brain on the formation, or vegetation, of the general mass of the blood; and, on the other, the deficiency of analyses of the blood in different diseases of the brain, with which the blood in apoplexy might be compared.

3. *Œdema of the brain.*—I have already mentioned several of the conditions under which the substance of the brain becomes infiltrated with serum. This occurrence is very frequent, and varies much in degree.

A slight amount of it may be recognized by the unusual moisture, and the brilliancy of a cut surface of the white substance, while its consistence remains unaltered.

A higher degree is marked by a soft pasty state of the texture of the brain, which becomes loose like pap, manifestly in consequence of infiltration of the tissue with a large quantity of serum. The œdematous white substance has a dull white appearance; in the atrophied brain of an old person it is of a dirty white, or fawn color.

When œdema reaches its highest degree, it breaks down the texture of the brain into a diffluent watery pap, from which serum escapes in large quantity. This so-called "white softening" (p. 270) is seen mostly in the neighborhood of the ventricles, and occasionally around tumors and inflamed portions of the organ. In such cases the rest of the brain also partakes of the œdema, though in a less degree.

Œdema of the brain may be developed rapidly, and quickly reach an advanced degree, or it may come on gradually and increase slowly. Instances of the former course are best marked in cases of acute hydrocephalus, in the cerebral substance surrounding the ventricles; and, as I have before remarked, it is seen occasionally in the cerebrum, near tumors, spots of inflammation, &c.

Chronic œdema of the brain is a frequent consequence of the most dissimilar chronic diseases, especially of phthisis; whether there be œdema in other parts, or not. It is very common in persons advanced in life, in whose atrophied brain it probably arises from the congestions resulting from vacuum within the skull. And it is equally common in insane persons, accompanying other marks of marasmus of the brain.

Œdema generally occupies the brain entirely, but not equally: it diffuses itself from some one spot, diminishing as it advances through the rest of the organ. The substance around the ventricles, when in the state of so-called "white softening," usually forms such a central spot (Concentrationsherd).

The enlargement of the brain to which œdema gives rise reaches its greatest degree when the amount of œdema is considerable, and the brain has lost but little of its volume by previous atrophy. In combination with the inconsiderable effusion which goes on into the ventricles, œdema is very often the principal cause of the swelling of the brain in acute hydrocephalus.

It destroys life, both by the pressure which this swelling exerts, and by the softening and breaking down of the substance of the brain in the spots where it is much advanced.

It is very probable, also, that in advanced age, after having slowly and gradually reached a certain relative degree of intensity, it occasions those common and unexpected deaths which simulate apoplexy.

As a general rule, hemorrhage does not take place in an œdematous brain.

APPENDIX.

Serous Apoplexy.

In considering the subject of œdema of the brain, a question already several times suggested, has again arisen, as to a mode of death, which is known by the name of "Serous Apoplexy." I have postponed answering it, till a more convenient opportunity; but now, having taken a survey of all the effusions of serum that occur within the cranium, to which, according to the present state of our knowledge, the production of serous apoplexy is, or might be, attributed, we are in a condition to enter into the subject.

The appearances usually mentioned as characterizing serous apoplexy are as follow: serous effusion into the sac of the arachnoid (especially a large collection, after the brain is taken out, at the back of the skull); infiltration of the meshes of the pia mater with serum; and a similar effusion between it and that part of the cerebral layer of arachnoid which stretches like a bridge over it; serous effusion into the ventricles; and withal, more or less distinct injection of the vessels of the pia mater with blood. To complete the picture, I must add, that the parenchyma of the brain is also infiltrated with serum. The essence of the whole condition would be an acute exudation of a large quantity of serum produced by congestion, and resulting in palsy of the brain.

The question then is, whether such a disease as serous apoplexy really exist, and whether it can be recognized in the body by the post-mortem appearances only, without reference to the symptoms attending the death of the individual? I would refer, on this subject, to what has been said on the difficulty of the question, at p. 259, and also to p. 291, where, when speaking of a disease similar to this in its final result of paralyzing the brain, viz., vascular apoplexy, I mentioned our limited power of determining, by what is found in the body after death, the influence of a disease in producing death (Lethalität). And I would now add that—

a. In the first place, we must exclude from consideration all cases in which hyperæmia originates from any pre-existing disease of the brain, such as inflammations, adventitious products, &c., as well as all acute effusions of serum resulting from them: all cases of hydrocephalus also, all secondary or subordinate (unwesentliche) chronic effusions of serum (those, for instance, which are produced by a vacuum within the cranium), cases of œdema of the brain in old and in insane persons, which destroy life by gradual palsy of the brain, and, lastly, all those effusions, which are proved by daily experience to take place without injuring the brain, and have no evident connection with the death, must be disregarded.

b. As it is a fact that substantive acute effusions of serum within the cranium do occur, their fatal influence will be "primâ facie" less doubtful, in proportion to their amount. They may supervene where gradual effusion has been already taking place. Œdema of the lungs presents the closest analogy to them.

c. The great frequency of chronic effusions, and their similarity, render it one of the most difficult of the duties of pathologist to recognize fatal effusions of this kind, and to determine that serous apoplexy has occurred in any particular case. The present state of our knowledge allows us to assert conscientiously that death has taken place in this manner only,—

When the effusions are considerable in some particular situation, and the membranes and brain are still distinctly vascular:

When the brain is swollen by the œdema; and, especially, when white softening exists in the neighborhood of the ventricles, but cannot be referred to previous hydrocephalus:

When it is not known that the individual has suffered from a previous affection of the brain:

When the morbid appearances which are frequently associated with it in other organs, especially in the lungs, such, for instance, as hyperæmia, hypostasis, œdema, are nothing but what the symptoms and the course of the agony show to have been merely consequences of the palsy of the brain, and cannot be looked upon as the actual mode of death, or as the cause of the congestion and exudation of serum within the cavity of the skull.

4. *Inflammation of the brain.*—Inflammation of the brain (encephalitis, the phrenitis of old writers) is not, on the whole, a rare disease, although it is so in comparison with other diseases of the brain: it is seldom found as a primary affection. It never occupies the whole of the brain, but appears in the form of circumscribed spots, which are sometimes very extensive.

It may be acute or chronic; it may produce disorganization of the brain, and reach its terminations by a rapid or a slower course. Some of its terminations have been most improperly regarded as chronic softening, that, for instance, which Durand-Fardel calls Cellular Infiltration.

When the disease occurs in the white substance, it presents in its various stages and degrees the following anatomical characters.

a. The diseased part is injected, streaked red, and swollen: the bleeding points seen on making a section of it are more numerous than natural, and not unfrequently its surface rises above the level of the incision. At the same time, the cerebral substance is unusually moist, and is already, in some degree, loosened or softened in its texture.

b. If the congestion continue, the redness not only acquires a darker hue, but also gradually discolors almost the whole texture of the part: for, besides being reddened by the injection, it is further discolored by dots and streaks which are situated outside the vessels, and result from numerous small extravasations into the tissue. Capillary apoplexy, as it is called, is thus found associated with inflammation. The swelling, moistness, and loosening of the texture, are all increased.

c. After this, exudation takes place, and, at the same time, evident modifications of the existing redness. A slightly plastic exudation pervades the cerebral texture uniformly, and, if not mixed with many of the red particles of the blood, it alters the dark redness of the part to a perfectly uniform and somewhat paler color. Exudations, which contain a large proportion of coagulable material, are still more remarkable: their coagulable part solidifies in masses of irregular shape and various size, but it is generally found, along the course of the vessels, in streaks or stripes of a yellow and greenish color. At the same time ruptures of vessels and hemorrhages take place, which are proportioned in number and extent to the violence (tumultuousness) of the process; and the fluid portion of the exudation percolates through the adjoining tissue in the manner before described. Hence there results a coloring, which is composed of several tints, associated and blended in the most different ways:—of dark red, from blood which has been extravasated at different periods; of paler reddening from infiltration of the cerebral tissue with the fluid part of the exudation; of yellow and green from the coagulated fibrin of the exudation; and of white from an occasional piece of cerebral tissue, which has escaped the process. The prevailing color, however, is the paler red of the cerebral tissue which is pervaded uniformly by the exudation.

At a later period, when this condition has existed for some time, the red color is further modified, by the coloring matter of both the extravasated and exuded blood gradually changing to a rusty brown, or to the yellow color of yeast.

d. The process of exudation, moreover, alters the cohesion of the diseased cerebral tissue. At first, as I have remarked, it is softened, but afterwards it becomes entirely disorganized. Its texture is broken up mechanically, partly by the extravasations, but principally by the exudation; and, very probably, it is also dissolved by the exudation. This is the condition for which the usual color of the diseased spot has procured the name of "Red softening of the Brain;" but the foregoing description of it, which has been drawn from repeated investigations, leaves no question as to its inflammatory nature. The softening is more marked, the greater the quantity of aplastic matter contained in the exudation.

During the stage of exudation (*c*), the inflamed spot might be described, especially by the softening, but further by the following characteristics: the substance of the brain is converted into a pulp, which is red, and variously shaded with dirty violet, brown, and yellow, or of a rusty or yellow color, like yeast; fragments of healthy brain, coagulated extravasations, and coagulated fibrinous exudation are scattered through the mass.

When the gray substance is the seat of the process, the anatomical characters are essentially the same; but the great vascularity and looser cohesion of this substance give rise to more of the redness from injection in the first stage, as well as to darker red coloring at later periods: the swelling, and loosening of tissue, the moistness, and the subsequent softening are also more marked.

Upon microscopic examination, the elementary tissues of the brain are seen broken up and dissolved, and amongst them the component elements of the different extravasations and exudations are met with in every stage of their progressive changes, both of degeneration and development. Amongst the latter are found, partially dissolved blood-corpuscles, shapeless masses of red, brown, and yellow pigment, single and conglomerate nucleoli in large quantity, globules and crystals of fat, nuclei, primitive cells, &c.

This description has been taken from cases as they ordinarily occur; but inflamed portions of brain are occasionally seen presenting much that differs from it.

a. Sometimes spots are found both in the white substance and in the masses of gray matter, which would be scarcely recognized as spots of inflammation: their nature can be determined only by a close examination, and from their analogy with similar inflammatory processes in other tissues. The redness resulting from injection of the part, is scarcely perceptible; other coloring is still more deficient, and the cerebral substance is loosened and softened to a uniform dull white pulp. On minute examination the texture of the brain is perceived to be broken down, and single and conglomerate nucleoli, nuclei, and pigment-molecules are found scattered throughout it. The inflammatory process, when at a moderate degree of intensity, has led to the effusion of a product containing very little plastic material.

β. Other inflamed spots, again, are found deep in the brain, and especially in the white substance, in which the softening, above described, is wanting, and hardness and resistance are the prominent characteristics. This does not appear to depend upon any induration (sclerosis) of the portion of brain which is involved in the inflammation, for it has undergone the same destruction when found in this condition as when softened, at least, in that most important particular, the forcible separation and breaking-down of its elementary structures; but its increased consistence arises from the coagulation of highly plastic fibrinous inflammatory products, and of the small extravasations which are present at the same time. The fact of such spots occurring is unquestionable; indeed, it would be surprising if they did not occur. They either go on to suppuration, in which case these coagulated products become converted into pus; or they change gradually into fibroid tissue, and form a cicatrix. In the latter case, there is never any softening throughout the whole course of the process, with the exception of the looseness of the cerebral tissue, that takes place at the beginning.

In the immediate neighborhood of the diseased spot, a slighter degree of inflammation prevails, and the appearances which it presents illustrate the commencement of the process. This part forms a ready medium for the advance of the disease upon any increase of the inflammatory action.

Changes of much importance occur in the cerebral tissue, still further from the diseased part. They are rarely absent; and thus a gradation of changes exists from the most diseased to the completely healthy structures. The outermost part is generally œdematous, or in a state of yellow softening.

The division of the brain in which the inflammatory process occurs, and, subsequently, even the whole brain, become permanently diseased; and this condition is more marked in proportion to the extent of cerebral substance included in the inflammation. It is forced out of its natural position by the accumulation of the essential, and of accidental, products of inflammation within it; it appears swollen and fluctuating, and presses upon the cranium, and on other parts of the brain, the latter being compressed by it: the convolutions of the cerebrum become flattened, and pressed against one another, and both cerebral substance and membranes are, in consequence, pale and bloodless. The division of the brain in which the inflammation occurs is of course œdematous, and more or less yellow softening surrounds the inflamed part itself, according to its extent, and the intensity of the process going on in it.

The usual site of the inflammation is the hemispheres of the cerebrum and cerebellum, the former being far more commonly affected than the latter. To speak more particularly, though it is met with somewhat frequently in the white substance of the cerebral hemispheres, yet, when we take into consideration the cases in which it happens at the periphery, there can be no question as to the greater liability of the gray substance to the disease. In that substance it is found both in the central masses of gray matter in the corpus striatum and thalamus opticus, and also on the surface of the hemispheres. And this distinction of an inflammation of the central gray masses and one of the gray matter at the periphery, is, like the same distinction in the case of cerebral hemorrhage, a matter of considerable interest. The resemblance of the two diseases, in respect to locality, is unquestionable, though Durand-Fardel endeavors to disprove it in the instance of the peripheral substance of the brain.

The inflamed spot varies in size from that of a bean or hazel-nut to that of a fist; it is sometimes met with so large as almost to occupy one entire hemisphere. In their early stage these large spots are not usually so extensive, they increase partly by the progressive enlargement of a previously smaller spot, and partly by several of such smaller ones coalescing. In the latter case considerable portions of brain, in a state of yellow softening, usually become involved in the diseased mass. The whole then generally assumes more or less of a rounded form.

The inflammatory action is generally confined to a single spot, though it is not very uncommon to meet with cases in which there are several distinct ones, in the same or in different portions of the brain. Very large spots often originate from the confluence of several smaller ones.

Encephalitis is far from being, as is generally asserted, a very acute disease; in nearly all cases its course is chronic. And this is true, even of traumatic inflammation. When it is the primary cause of death, we may trace this result either to the large extent of brain destroyed, or, as is more commonly the case, to the pressure and anæmia produced by the swelling of the brain, when œdema and yellow softening have come on around the inflamed spot. It destroys life in the same manner sooner or later, by its terminations and consequences; and last of all, by general marasmus, by congestions, especially in the lungs, and hypostatic pneumonia, by sloughs on the sacrum, &c.

a. Encephalitis may terminate in *resolution*, but only at the beginning of the process, and when it is very slight in degree.

b. It results also in *atrophy*,—in secondary atrophy. The principal part of the products of the inflammation is absorbed, together with so much of the injured cerebral tissue in which they are contained, as is incapable of being restored to a healthy condition; while a small portion of the products undergoes a change of structure. This termination is in itself one of great importance; but it also requires more especial notice, because its true character as a termination of inflammation has not hitherto been recognized, and it is looked upon by observers as the true softening, and, indeed, as the highest degree of softening of the brain. (See Andral and others.) In the seat of inflammation we find an extremely delicate, and irregular network, of a white, grayish, or very pale yellowish-red color; sometimes it is soft, sometimes pretty firm: its interspaces are filled with a fluid, like an emulsion or lime-water, flocculent, white and turbid. Upon minute examination, this delicate network is found to consist of a fine cellular tissue at various stages of development, and of a few vessels. The fluid contains a quantity of separate and conglomerate, elementary corpuscles, fat-globules of different sizes, and a little amorphous pigment, which is not discernible by the naked eye. It constitutes the second stage of Durand-Fardel's chronic softening, and is called by him *cellular infiltration.* Both it and the following latter stage of the same morbid process occur only in the white matter, or only in those of the cerebral ganglia which contain much white matter. Accordingly, I have observed it unquestionably in the corpus striatum, but, like Durand-Fardel, have never discovered it in the peripheral gray matter. Gradually the fluid becomes clearer, and at last we find at the affected spot a cavity, lined by a layer of delicate cellular tissue, and usually honeycombed, or traversed in different directions by slender filaments of cellular tissue, and containing a clear serous fluid. The cerebral substance immediately surrounding the cavity appears slightly condensed,—sclerosis.

Even very extensive inflammations terminate in this manner. There is no question that the cavity, just spoken of, may gradually diminish, and at length close, like the apoplectic cyst,—for which it is not unfrequently mistaken. The diagnosis between them (which I will mention at once) is often very difficult, and sometimes cannot be made without referring to the early symptoms of the disease. The difficulty arises, on the one hand, from the fact, that high degrees of inflammation are, at first, always complicated with hemorrhage; and, on the other, that inflammation often supervenes upon hemorrhage. The apoplectic cyst generally has its well-known rusty-brown or yellowish lining; but it loses, in the course of time, nearly all its coloring matter. The cavity left after inflammation usually has no such lining; but sometimes, when considerable hemorrhages have taken place, the inner membrane of this cavity also is colored with rusty brown, or yeast-like, yellow pigment. If a more intense inflammation should have occurred around the seat of hemorrhage in the brain, and produced the so-called cellular infiltration and its consequent cavities, the apoplectic cyst can usually be distinguished in the midst of them, by its colored lining. It may, however, have almost lost this lining, or the other surrounding cavities may also be colored with a coating of pigment.

c. Inflammation may terminate in *induration*, sclerosis. At the seat of inflammation, there remains a white or dirty white, rounded, elongated, or irregularly branched cicatrix; or, in a few cases, a callous, grayish, semitransparent substance. The cicatrices which are occasionally found scattered in considerable numbers throughout the brain, in persons subject to convulsions and epilepsy, are most probably examples of the former kind. They consist of a compact mass of elementary molecules, traversed by a few delicate fibrils like cellular tissue. The termination in induration combined with a growth of fibroid tissue, and condensation, may be noticed around tubercle, abscesses, &c.

d. When the process terminates in *suppuration* and *abscess*, the products of the inflammation become converted into pus; and the other tissues involved in its area, the extravasated blood and cerebral substance, with its vessels, perish (necrosiren) and disappear.

The recent abscess is a rounded cavity, irregularly hollowed out of the parenchyma of the brain. Its walls are composed of suppurating cerebral tissue, sloughing shreds of which, soaked in pus, hang inward into the cavity, while all around the brain is in a state of inflammation, —of red softening. The more distant cerebral substance is œdematous; and very often the part encompassing the area of red inflammation, is found in a state of yellow softening, which has occasioned death. The diseased division of the brain, and sometimes the entire organ, is swollen, anæmic, fluctuating, &c., in degrees proportioned to the size of the inflamed spot, and the activity of the processes going on around it. The pus contained in the abscess is thick and greenish, and has an extremely fetid phosphorescent odor.

By continued inflammation and suppuration, the abscess advances beyond the bounds of the original inflammation. Sometimes it enlarges uniformly on all sides; but it generally does so in one direction only, and in the form of a sinus.

When an abscess thus enlarges, and the process of ulcerative inflammation extends to other structures, such as the membranes of the brain, the cranium, &c., an outlet may be formed, and the matter discharged. It is, however, a necessary condition, that the inflammation of the inner membranes be limited, and do not spread out into an extensive meningitis. Such outlets may be formed at various parts of the skull, and the matter be discharged either directly outwards, or into natural cavities and canals which convey it away. In this manner pus is discharged from an abscess of the brain, through the cribriform plate of the ethmoid bone into the labyrinth and cavities of the nose, or through the tympanum and meatus auditorius externus. In the latter case, the roof of the tympanum or the wall of the mastoid cells is destroyed by caries, and the matter finds its way into the tympanum; from whence, after ulceration of the membrana tympani, it is discharged. Such a purulent discharge bears the name of otorrhœa cerebralis, inasmuch as the matter, though coming out at the ear, proceeds, in part at least, and for some time, from the brain. Albers considers that the priority of the affections of the brain and ear may be reversed, and sometimes one, sometimes the other, may be the original disease: while, in a third case, both may arise together, from a common cause.

The rupture of an abscess of the brain into the ventricles is always rapidly fatal.

Although abscess in the brain is usually fatal, yet patients very often live on with it for a considerable period, and sometimes without presenting any clear evidence of its existence. In such a case the abscess has become encysted, and is even capable of being completely healed.

The abscess becomes enclosed in a firm capsule, by the conversion of the granulating layer on its inner surface into a dense, cellulo-fibrous (fibroid) membrane, while the adjoining layer of cerebral substance becomes condensed, and like a cicatrix.

The inflammatory process may be renewed in the walls of an encysted abscess; or œdema, or yellow softening, may take place in its neighborhood, and, in either way, death very frequently ensues. But as I have said, the abscess may heal. The pus is then partly absorbed, the remainder of it becomes inspissated, and forms a chalky residuum; and the capsule enclosing it contracts equally, and at length altogether wastes.

Every inflammation of the brain may terminate in suppuration; but it is more especially those inflammations which are produced by wounds, and concussion of the brain, that take this course.

The destruction of brain, and the loss of its substance resulting from the inflammatory process, give rise to a remarkable consequence. The nerve-fibres which are involved in the inflamed spot become atrophied, more or less plainly, according to circumstances. And, after repeated attacks of inflammation, the whole brain becomes also tough and wasted, in the same manner as after the frequent occurrence of cerebral hemorrhage.

Encephalitis may be a spontaneous disease, or it may come on as a traumatic affection from a wound, or concussion of the brain. In the former case, it is sometimes the primary disease; but very frequently it is secondary, or consecutive. Thus it is often developed in the neighborhood of malignant deposits, around the bed of hydatids, or about foreign bodies which have lain for an indefinite period in the brain without producing injury, &c. When it happens as the primary disease, it is very frequent in persons advanced in life, and in the aged; it is, however, far from being peculiar to them; no period of life, not even childhood, is exempt from it, and I have unquestionably observed it in the immature fœtus. It seems to depend, principally, upon a liability to passive congestions, for it is common in old and much-reduced persons, and is not uncommon in the period of debility following the exhausting acute dyscrasiæ, especially typhus. Hypertrophy, or dilatation of the heart, is not unfrequently also present, and may favor the production of the disease.

After these remarks on encephalitis in general, some special notice is required of inflammation of the periphery of the brain. The same reasons which make peripheral apoplexy important, render this so too; and, indeed, peripheral inflammation is, if possible, the more serious, from its not unfrequently occupying a great part or even the whole of a hemisphere, and often giving rise to secondary imbecility.

It presents the same general appearances as inflammation of the gray matter elsewhere. Even when slight in degree, and when but just commencing, it may be clearly recognized, on exposing the surface of the brain, by the dark-red coloring and swollen state of the inflamed part, and by large pieces of cerebral substance adhering to the pia mater, and coming away when the membranes are peeled off. Higher degrees of it are generally combined with inflammation of the pia mater itself; and sometimes it is not confined to the peripheral substance of the brain, but it involves also the pia mater, the arachnoid, the dura mater, and even the cranium, as is proved by the adhesions formed between those different structures, and by their increase in size.

Peripheral encephalitis is mostly situated in the gray substance of the convolutions on the convexity of the cerebral hemispheres; it is less frequent at the base, and still less on the cerebellum. It is also generally remarkable for its great extent.

Peripheral inflammation terminates in *resolution*, or in a state of *looseness* of the outer layer of the cineritious structure, in which it clings to, and may be stripped off with the pia mater.

Or it may terminate in *atrophy*, and *absorption*. The cineritious layer then becomes thinner, and either whitened, or of a dirty fawn or yellow color; or else it is completely removed, and then the white substratum of the convolutions is laid bare, and appears condensed and callous. My observations correspond with those of Durand-Fardel, in having never found in the periphery of the brain, that condition of parts which he has named cellular infiltration.

The last termination of the disease is *induration*. The convolutions are found tough and callous through their whole thickness; they are also paler than natural; and the pia mater covering them, having become condensed and tendinous, adheres closely to their surface.

I have already spoken (at p. 298) of the so-called yellow plates, which Durand-Fardel attributes to chronic softening of the surface of the brain, and of the ulceration, as it is called, of the surface of the brain, which is associated with it. The former are the remains of the extravasation: and the so-called ulceration is produced by absorption of so much of the substance of the brain as had been broken up by the hemorrhage. Both appearances certainly may be found combined with some remains of inflammation, especially with cellular infiltration in the adjoining white substance.

A later and a most important consequence of these terminations of peripheral inflammation is a gradual *atrophy* in the interior of the brain, which, when the original process has been very extensive, involves the entire hemisphere. It is accompanied by induration and a dirty white discoloration of the fibrous substance. These appearances are sometimes so marked that the whole disease might be taken for an original and congenital arrest of development; and the more uniformly the several parts of the hemisphere are wasted, the more likely is the mistake. Moreover, the atrophy is usually concentric, so that the lateral ventricle is diminished in size. The vacuum is filled up by thickening of the membranes, and an effusion of serum around the hemisphere; and in the case of the cerebrum, by an enlargement of the opposite hemisphere, which is due to the dilatation of its lateral ventricle.

5. *Metastasis.*—In some cases, in which the blood is poisoned by containing a large quantity of purulent matter, as, for instance, in the purulent diathesis, or pyæmia, of lying-in women, the brain becomes the seat of metastatic purulent deposits. Under these circumstances, other organs and tissues are always found to have undergone the same morbid process. A marked characteristic of these deposits in the brain is their great number. They occur in all parts of the organ; but they are most commonly found deep in the substance of the cerebral hemispheres. As in other parts of the body, they are developed from a circumscribed dark-red plug or core, and they form collections of pus or of ichor, the usual size of which ranges between that of hempseed or a pea and that of a bean.

6. *Softening of the Brain.*—The most prominent feature of softening of the brain, or encephalomalacia, is the alteration of consistence: but the disease results from other fundamental changes, and is treated of amongst the diseases of texture only for want of a more suitable place. Our previous investigation of certain examples of softening will have prepared us for the consideration of the whole subject, and we shall now, with regard to them, be referring only to what has been already asserted.

Notwithstanding the labors of many older and more recent observers, the anatomical diagnosis of softening of the brain is far from being either clear or complete. Without doubt, this deficiency has principally arisen from their neglecting to distinguish accurately the several forms of the disease. And it has been needed also that observations should be extended to analogous processes in other organs, especially to inflammation, and to that softening, disruption, and solution of tissue which characterizes inflammation wherever it occurs, as well as to its terminations in other structures also. Moreover, the disease has, from want of material, been insufficiently observed in its several stages; and, lastly, a lack of special observations supported by chemistry, especially of observations on yellow softening, has contributed to the same result.

Softening of the brain occurs under such totally different forms that it cannot be treated of as one general disease. For the same reason it is impossible to decide the question, whether it be of inflammatory nature, that is, produced by inflammation or not; a question which is answered in the affirmative by one large party, and in the negative by another equally large.

There are three essentially different forms of softening of the brain: two of them have been already spoken of; but, in order to complete the account of the disease, they must be again brought forward.

a. The first form is that which is met with in hydrocephalus (p. 269), and œdema of the brain (p. 304), as *white*, or hydrocephalic softening.

It consists in a loosening and subsequent laceration of the cerebral texture by an interstitial exudation of serum. Like œdema in general, it sometimes takes place without any inflammation, at other times it is unquestionably so far inflammatory that a certain quantity of a coagulable blastema, capable of assuming an elementary organization, is poured out with the serum. Examples of it are furnished in the more or less acute forms of œdema, which occur in the neighborhood of spots of inflammation, and more especially in the œdema which accompanies acute meningitic hydrocephalus, and destroys the tissues around the ventricles of the brain. In such cases of softening, the characteristic products of inflammation may generally be discovered with the microscope in the diffluent cerebral mass.

It does not appear to me necessary to suppose (with Paterson), that the brain is naturally hygrometric, in order to explain the occurrence of such a softening in the neighborhood of the ventricles, in cases of acute hydrocephalus; I have made frequent experiments, but have never found imbibition to produce a softening of the cerebral tissue, in any way resembling that which takes place in hydrocephalus.

Nor can I adopt Fremy's view (which will be taken into consideration, together with the subject of yellow softening). For white softening has, apparently at least, no connection whatever with putrefaction, and it is certain that it often exists for a long period as chronic œdema of the brain, and yet the serum is found to have caused no maceration or decomposition of the cerebral substance.

It is quite different from yellow softening, and has no analogy whatever with the process of softening in the stomach.

Its essential character, viz., that of destroying the cohesion of the brain, was recognized by Laennec.

b. The second form is that which has been described in the article on Inflammation, as *red* softening (p. 307), and in some few cases as a softening, marked by *dull white* discoloration (p. 308). There can be no doubt of its inflammatory nature: and in the trifling amount of the discoloration, *i. e.* the whiteness of the softened tissue, the latter variety shows its alliance to inflammatory œdema. The softening results from the cerebral tissue being broken asunder and dissolved by the exudation.

This class includes, moreover, that condition which has been described (p. 310) as a termination of inflammation of the brain, as the termination in atrophy or absorption, or the so-called cellular infiltration.

All these softenings are found, as has been noticed in the separate descriptions of them, not only as primary and substantive, but also as secondary and symptomatic.

c. The third form is the *yellow* softening, which has hitherto been only occasionally mentioned. It is on every account a remarkable disease of the brain, and yet, singularly enough, it has received but little attention from observers until very recently, and is only cursorily mentioned by them: it is, however, the instance of softening that best supports the numerous opinions of German and French physicians who oppose the theory of its being of an inflammatory nature, especially those opinions according to which it is a disease *sui generis*, a specific alteration of nutrition, &c.

Yellow softening, like inflammation, never attacks the whole brain at once, but occurs as a primary and idiopathic disease in pretty sharply circumscribed spots.

At a spot which may vary in size, but which is scarcely ever larger than a hen's egg, the cerebral substance appears converted into a very moist, tremulous pulp, of the yellow color of straw, or sulphur, and not unlike brine (sulzeähnlich): when cut across it rises considerably above the level of the section; and it presents to the naked eye no trace of natural cerebral structure. The transition from the diseased to healthy structure is somewhat abrupt, passing through a thin layer of cerebral substance in which the disease is less advanced; the texture of the brain immediately around is found to be comparatively normal.

These are the appearances in a well-marked case; but there are different degrees of the affection as may be seen in the immediate neighborhood of a part which is thoroughly disorganized. They are distinguished by the different amounts of discoloration, *i. e.* of saturation with color, and by the degree in which the texture is infiltered and disorganized.

In slight degrees of the disease, the portion of brain affected is of a dull white color, inclining to yellow, and is evidently moister and softer than natural.

When it has reached a higher degree, the moistening and softening are more considerable; but a further circumstance may be noticed, which is important, both generally and specially: it refers to the degree in which the cerebral texture is disorganized. When a section of the part is made, or portions of it are squeezed in the course of the examination, a considerable quantity of clear, or nearly clear, yellow, thin fluid gradually oozes out; or it may collect in fissures, which form spontaneously: it leaves behind large coherent masses of softened, but still of white cerebral substance.

The highest degree is characterized by the complete or saturated yellow discoloring of the diseased part, and by its containing the briny pulp before alluded to. Even under the most favorable circumstances, the yellow fluid yielded by this pulp does not strain off from the cerebral mass, it always brings away a sediment composed of delicate shreds of broken-down cerebral tissue.

There is neither vascularity nor reddening in or around the seat of disease, though sometimes, indeed, small extravasations give a dotted or streaked appearance of redness to the part (ecchymosis).

The presence of a spot of softening occasions more or less turgescence of the diseased portion of the brain, or of the whole organ. The swelling is due partly to displacement, and partly to congestions or œdema of the brain. It is remarkable how much swelling of the brain yellow softening produces. Spots of the size of half a cubic inch, or of a nut, give rise to quite a disproportionate turgescence. Though this may depend in great part upon œdematous infiltration of the cerebral mass, yet the other cause, viz. the displacement, is not to be overlooked; for the much greater space occupied by the softened portion than by that which is healthy, is quite palpable. How great this enlargement is, and the tendency of the diseased portion of the brain to expand, is manifest also from the appearances observed when a section of the part is made.

The most common seat of yellow softening is the cerebrum; it is less usual in the cerebellum, and is scarcely ever found in the pons and the other structures at the base of the brain. It attacks both the fibrous structure and the central masses of gray matter, but it is extremely rare in the periphery of the brain, and when it does occur there, is almost always secondary. There is no essential anatomical difference between yellow softening of the gray matter and that of the white.

The affected spot varies in size from that of a bean to that of a walnut or a hen's egg: I have as yet seen no examples of primary and uncomplicated yellow softening which exceeded those dimensions. There is generally only one spot, and its shape is round.

Yellow softening is met with both as a primitive idiopathic disease, and as secondary and symptomatic. The foregoing remarks apply principally to the former kind. The latter is the more frequent. It accompanies inflammation, hemorrhage, and adventitious products of the most different kinds in the brain, affecting the cerebral substance immediately around them. In cases of inflammation, it not only encircles the diseased spot, but is seen also at different parts in its interior; for the portions of cerebral tissue which sometimes remain uninjured within large areas of inflammation may afterwards be attacked with yellow softening.

It is sometimes noticed in the neighborhood of the ventricles, forming the central spot from whence acute œdema radiates in cases of acute hydrocephalus.

The cerebral substance immediately around an apoplectic clot, or adventitious product, is occasionally found in a state of yellow softening; but far more commonly there is an interspace of inflamed tissue (red softening) between the central disease and the yellow softening.

The diseases of the brain which have just been mentioned are the usual pre-existing and the most important diseases with which yellow softening is found combined.

Yellow softening appears to be always and rapidly fatal: and, in those diseases of the brain which have been mentioned, it is in fact the immediate cause of the fatal result. According to my observations it runs a rapid course, especially in its secondary form, though no doubt cases of idiopathic yellow softening may last for a longer time. There does not appear, *à priori*, to be any reason why these spots should not heal; they might do so in the same manner as the apoplectic cyst, or inflammation; but, as yet, we have no facts to prove it.

Yellow softening, primary as well as secondary, may occur at any period of life; but, under both its forms, it is most common in middle and advanced age.

Its nature is still quite problematical.

It has been remarked above, that in a slight degree of softening, a thin yellow fluid oozes from the diseased portion of brain. This fluid, on minute investigation, presents a number of broken, extremely varicose, primary tubes, with their contents, of swollen blood-globules, very transparent fat-globules of various size, and some yellow amorphous pigment. I have repeatedly tested the fluid, and been convinced that it is somewhat intensely acid.

The first question which arises is, whether yellow softening be of inflammatory origin. The following points must be considered in the attempt to solve it.

a. At no stage of the primary form of the disease, from the earliest period at which it can be recognized as yellow softening, is any injection or reddening perceptible; nor is there any vascularity worthy of notice in the parts adjoining or more distant from it, if we except the congestions, which, from the existing œdema, we may infer to have previously taken place.

b. The same remark applies to the secondary softening. This form either immediately surrounds a spot of apoplexy or an adventitious product (in which case, as in the primary, it is entirely free from vascularity and reddening); or it skirts a spot of inflammation, or some inflamed cerebral substance which itself surrounds an apoplectic spot or adventitious product. The line of boundary between the inflammation and the softening is, however, sharply defined, and no injection or reddening extends beyond it into the region of the latter. It may even surround the rarer kind of dull white inflammation.

c. It does not contain those products and elementary formations which are usually found in inflammation.

d. The yellow color is certainly not dependent on the presence of pus and pus-cells; nor is the proper color of the fluid that of pus; it acquires its yellow puriform tint from being mixed with cerebral tissue.

The theory, therefore, which ascribes the origin of yellow softening to inflammation appears to be quite untenable.

Blood-corpuscles, and the pigment already alluded to, have as little to do with the production of the yellow color as purulent matter. Especial care must be taken not to commit the common error of confounding the color of yellow softening with the rusty-, yeast-, or ochre-yellow tints of the cerebral substance in cases of apoplexy and inflammation, which undoubtedly depend on the coloring matter of the blood. The color in yellow softening entirely differs from these; and, indeed, cannot arise solely from the coloring of the blood, for the fluid contains far too small a quantity of blood-corpuscles, as well as of the amorphous pigment, to account for it.

It is remarkable, that yellow softening of the brain never gives rise to any inflammatory reaction around it.

My own impression is, that yellow softening is founded in a chemico-pathological process.

As Couerbe's view, which was suggested by the equivalence in composition, or the isomerism, of the Eléencéphol of the brain with another of its constituents, the Céphalote,[1]—a view which was adopted by Magendie,—must be rejected as a mere ingenious combination; so neither can Fremy's theory be accepted, which was drawn from the process of putrefaction in the brain. But it must be admitted that, whilst yellow softening does not exhibit the remotest similarity to the decomposition of the cerebral substance in the putrefactive process, much use may be made of Fremy's views, and that they have given a direction to future investigation which is full of promise.

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[1] [See Magendie's Léçons Orales, t. i. p. 159.—Ed.]

He considers the brain to consist of cerebric acid, either free or combined with soda and phosphate of lime, of oleo-phosphoric acid, free and in combination with soda, of olein and margarin, of small quantities of oleic and margaric acids, of cholesterine, water, and a substance like white of egg, in the proportion of 7 parts of albumen, 5 of fatty matters, and 80 of water. The oleo-phosphoric acid, which, like the olein, is usually yellow, is very easily acted on (sehr veränderlich), and separates readily under slight influences into phosphoric acid and olein. Thus it decomposes at an ordinary temperature, when it comes into contact with water; and decomposing animal matters give rise to a similar change in it. Now, what first occurs in putrefaction of the brain is this decomposition of the oleo-phosphoric acid. But the process does not stop here; for the albuminous matter, also decomposing, sets up a further decomposition in the olein, and genuine saponification is the result—a conversion into oleic acid, and a combination of that acid with ammonia. Fremy thinks that this is the process which goes on in softening of the brain,—that it is, in fact, a genuine putrefaction of the brain. Although, as I have already said, I cannot discover any of the phenomena of putrefaction in the process of yellow softening, yet the liberation of an acid, —the phosphoric, and especially one or more of the fatty acids,—may be conjectured to be one of the most important phenomena in yellow softening. The conjecture is supported by the very decided acid reaction of the fluid contained in the softened spot.

A second question which arises has reference to the proximate palpable cause of the process. Considering how commonly yellow softening occurs both around a spot of inflammation, and in those portions of brain within it which are uninjured by the exudation, that it is also developed outside the halo of inflammation which surrounds apoplexy and morbid growths, and even immediately around these growths themselves, we may infer that obstruction of the vessels in the inflamed part, or impermeability of them from pressure, and the consequent impeding and interruption of the circulation in a portion of the brain, may be one considerable predisposing cause. And then, under certain circumstances, the contact of the cerebral tissue with extravasated blood in different stages of metamorphosis, and with the products of inflammation, might give the first impulse to the occurrence of yellow softening.

It appears, then, that yellow softening, and it alone of all the examples of softening of the brain, may be ranged side by side with softening of the stomach. In both of them alike, the possibility of any inflammatory reaction in the diseased tissue is absolutely precluded.

7. *Induration of the brain—Sclerosis.*—The consistence of the brain is liable to increase under very different circumstances, and in every degree, from that which is imperceptible to that of a leather-like or fibrocartiliginiform hardness and resistance. In the slighter degrees, there is no evident or decided anomaly, and it is most probable that they mainly depend on a diminution of the quantity of water in the brain; but in decided cases it arises from atrophy, shrinking, and change of structure. The more important indurations, therefore, must be classed among the diseases of the texture of the brain.

Induration is either total or partial: it affects the whole brain equally, or some single, larger or smaller, part of it only. The partial indurations are generally distinguished for the great degree of hardness they attain, and are known as a callous state or cicatrix of the brain.

The minor degrees of increased consistence, that, for instance, which is commonly selected for the study of the fibrous arrangement of the brain, are best marked after great and exhausting exudative processes, such as peritoneal exudations in puerperal women, or in the course of ileo-typhus, typhoid fever, and acute exanthemata, especially of scarlatina. And here, in opposition to Gluge, I must expressly state, that such a degree of condensation of the brain is the rule in typhus; while, on the other hand, decided softening, which, in fact, is nothing more than œdema of the brain, is certainly common later in the disease, in the train of evils connected with the disordered states of the system which follow typhus.

In poisoning by sulphuric acid (Otto), though I have had abundant experience, I have met with no instance in which the consistence of the brain was increased. In poisoning with lead, on the contrary (Andral, Tanquerel), though the consistence of the organ was sometimes increased, and, in a few cases, a high degree of it was combined with shrinking of the brain, there was much more commonly an unnatural softening resulting from œdema.

More advanced degrees of increased consistence, those in which the cerebral mass becomes tough like leather, accompany atrophies of the brain, as well those which are total, as, and more particularly, those which are partial. The most marked example of them is that which attends the partial atrophies resulting from a previous change of texture, apoplexy, or inflammation (p. 288). In such examples, circumscribed dense cicatrices are found in the parenchyma of the brain.

Induration in its highest degrees presents a fibrous, cartilaginous, or scirrhous hardness, and results from a serious disease of texture, especially from infiltration of the cerebral substance with cancerous matter. The disorganized portion of brain appears to have nearly or entirely lost its natural texture; and, in the latter case, the cerebral substance at length disappears in the foreign mass. (Compare "Cancer of the Brain.")

8. *Adventitious growths.*—Although there are some forms of adventitious products which are rarely seen in the brain, yet there are others which are comparatively frequent: so that this class, on the whole, supplies an average number of the diseases of the organ. Among the most frequent are tubercle and cancer.

Their importance is proportioned to their size, to the vascularity and looseness of their texture, and their consequent liability to swell, to the rapidity of their growth, to the degree in which they give rise to congestions in their own immediate neighborhood, or in the whole brain, whether it be by acting as foreign bodies, or in consequence of the change of material going on in them, &c. They lead to displacement of the brain and pressure; and thus interfering with the injection of its vessels, they produce anæmia in it; they cause congestions also, and swelling of the organ, and finally hypertrophy, œdema of the brain, and hydrocephalus, inflammation and yellow softening in the neighborhood, &c. And so death occurs, sometimes gradually, at other times in a rapid and unexpected manner.

a. Fatty tumors.—This disease is very rare in the brain: when it does occur it will be found to spring rather from the tissue of the pia mater, and lining membrane of the ventricle than from the cerebral substance. This is especially true of the genuine lipoma. Examples of encysted cholesteatoma (adipocire-like tumors) are also met with.

b. Cysts.—Though these growths are rare, they are less so than the lipoma; the only form in which they occur is that of the sac of the acephalocyst. The mother-sac of acephalocysts in the brain generally has such remarkably thin coats, that it may be easily overlooked; and a single acephalocyst, when it completely fills the mother-sac, might readily be regarded as the sac itself. The cases which I have met with have all been of that kind, namely, a mother-sac, completely filled with a single acephalocyst. Other observers have seen cases in which the acephalocysts were more numerous, and different museums contain examples of them.

Neither simple nor compound cysts seem ever to be formed in the brain.

c. Fibroid structures.—Tissues of this kind, as well as newly-formed cellular tissue, are met with in various stages of development, in apoplectic and inflammatory spots, in the wall of the apoplectic cyst, and of a healing inflammatory spot, and in their cicatrices, in the membranous wall of an abscess, or of a tuberculous cavern in the brain, in the capsules of many of the adventitious products, &c.

Fibrous tumors are very seldom found in the brain: there are many cancerous growths which bear a deceptive likeness to them, and which are very commonly mistaken for fibroid growths.

d. Chalky concretions.—These concretions are found in the form of circumscribed accumulations of a dull white chalky powder, or of a yellow mortar-like, or coherent solid mass. They are generally enclosed in a cyst, which may be thin or thick. They consist essentially of the thickened and cretified contents of an abscess, of cretified tubercle, of the chalky contents of the bag of a dead cysticercus, or the like.

e. Tuberculosis.—Tubercle is the most common of the adventitious products in the brain, and it is frequent in comparison with other diseases. Tuberculosis presents several peculiarities in the brain.

The *number* of tubercles is usually very small, one or two being met with in most cases, and more rarely three, four, five, or a few more: some extremely rare exceptions do occur, in which twenty and more are found. When there is a small number of tubercles, each separate one acquires a considerable size; but when they are more numerous, no single one becomes much larger than a hemp-seed or pea. There is also a further difference between tubercles in the brain and those in other organs, that the former are usually found not aggregated together, but widely apart from each other.

Tubercle in the brain generally attains a considerable *size*, and is larger in proportion as the number of deposits is small. Its volume varies from that of a millet-seed to that of a hen's egg, or more; the most usual size is that of a hazel-nut or walnut.

It appears, therefore, that tubercle in the brain presents considerable difference, in respect to the size and number of the deposits, from the same disease in other organs, especially the lungs. These conditions of its development correspond most nearly with those by which tubercle is developed in the lymphatic glands,—with which, moreover, it is often found combined.

There is no peculiarity in its *form*, by which tubercle of the brain can be distinguished from large tubercular masses in other organs. It may be round, or may assume the most irregular, branched, or lobular shapes.

Every part of the brain is occasionally the *seat* of tubercle. It is very common in the cerebrum, and less so in the cerebellum; while it is rarely found in the pons, and still more rarely in the medulla oblongata. As a general rule, it is deposited in or near gray substance: its usual situation, therefore, is the cerebrum, either near the periphery, or more deeply amongst the gray portions of the corpora striata and optic thalami. The corpus callosum, fornix, septum lucidum, and crura, scarcely ever contain any.

Tubercle of the brain, when it is situated near the surface, and is of large size, frequently encroaches on the superficial layer of cerebral substance, breaks through it, and fixes in the tissue of the pia mater. By giving rise to exudations in this membrane and the arachnoid, it sometimes becomes adherent to the inner surface of the dura mater, and though in reality originating in the brain, it is then very liable to be looked upon as tubercle of the membranes.

The usual condition in which tubercle is found in the brain, is that of an adventitious mass of the size and form already mentioned, of a yellow or yellowish-green color, of the consistence of lard or cheese, and firm, but easily lacerable; when situated deeply in the brain it is often fissured, and spreads out in various directions. The greatest part of the mass, and sometimes apparently the whole of it, are thus constituted. It is generally, howener, surrounded by a considerable layer of pale reddish vascular substance, which is also firm, and which when minutely examined, is found to consist of nuclei, of cells in different stages of development, and isolated shreds of cerebral tissue. In some parts it often appears paler than elsewhere, of a dull reddish-white or white color, dense and resistant like a cicatrix, and in fact, in course of conversion into a fibroid callus. Outside this layer an extremely delicate, moist, and jelly-like cellular structure connects the tubercle with the surrounding cerebral tissue; but its texture is so slight that the whole morbid mass may be easily loosened and turned out of the brain. This stratum further contains, scattered mostly through its inner part, some small gray or grayish-yellow tubercles, which occasionally unite with the great central mass. Although this cellular stratum surrounding genuine tubercle in the brain, is sometimes very thin, yet it is scarcely ever absent; it is the product of a moderate inflammatory process tending to induration, and corresponds to the like process which occurs around large masses of tubercle in other tissues.

As tubercle in the brain is generally discovered in the form depicted above, bearing marks of a previous process of softening, it may be asked, whether it has no crude stage in that organ, that is, a stage of gray translucent granulation, or whether it is at once secreted as the yellow lardaceous, or cheesy tubercle. There are some rare cases which prove that tubercle in the brain does, in part at least, commence in the gray, translucid form, for portions of a tubercular mass are sometimes found in that state. In any case, however, it may continue for a short period only in that form, and soon pass into the stage of the yellow cheesy tubercle.

Tubercle is usually fatal when it occurs in the brain, before the further changes of which it is susceptible, viz., softening and cretification, can occur. The former, however, is not extremely rare. When the tubercle is entirely *softened*, its place is found occupied by a cavern, mostly of a spherical form, which is enclosed in the peripheral vascular reddish stratum before described, and has the characters of an encysted abscess. The interior of this stratum is in a state of suppuration; and sometimes secondary deposits of tubercle, which may also soften, are found in it. As, after some time, the early characters of the tubercular matter may disappear, it is often very difficult to distinguish the abscess which has originated in tubercle, from that which has resulted from inflammation; and it is only by the concurrence of tuberculosis in other parts, and more especially in the brain, that the diagnosis can be rendered certain.

Cretification of a tubercle in the brain is one of the most uncommon metamorphoses which tubercle ever undergoes; but yet it does occur in some cases, not only in one, but even in several extensive tubercles.

Tuberculosis is a chronic affection in the brain. An acute form of the disease is unknown in that organ.

Brain-tubercle, as well as tubercular abscess, may exist for a long time before the changes which it occasions, either in the whole brain, or in the adjoining cerebral substance lead slowly or rapidly to a fatal termination. A general description of these changes is given in the Introduction.

It must also be remarked that death may ensue from inflammation of the membranes with tubercular exudation, and from acute tuberculosis of the membranes at the base of the cerebrum, combined with acute hydrocephalus. Both these are processes which originate in tuberculosis of the brain.

Tubercle in the brain, like tubercle generally, may occur at any period of life; but it is notorious that in this situation it is especially a disease of childhood and youth. In some very rare cases it is the only instance of tubercle in the organism, but, as a rule, it is combined with tuberculosis of other structures. Its most frequent combination is with tuberculosis of the absorbent glands, which prevails most at the same period of life. Next in order of frequency to the glands, is its association with tubercle in the lungs.

f. Cancer of the brain.—There is no organ in which growths of a cancerous nature occur in such number, and in such variety of internal elementary structure, as in the brain. I must refer the reader on this subject to the general remarks on cancer, and the morbid products allied to it.

They are met with either infiltrated through the cerebral substance, so that the transition from diseased to healthy structure is gradual and insensible; or, as is far more common, in the form of an independent and abruptly circumscribed tumor, which is enclosed in a delicate areolar and vascular investment.

The infiltrated cancer generally involves a very considerable portion of the brain, while the tumors attain various dimensions; sometimes equalling a hen's egg in size, sometimes exceeding the fist. They are generally spherical; but when their texture is soft, they readily accommodate themselves to any firm adjoining structure; and those even which are dense and tough, when situated at the periphery of the brain, become flattened on their surface, or excavated like a navel. They sometimes advance from the brain to the membranes, and through them to the cranium.

The infiltrations sometimes render the diseased portion of brain tough and firm, like a cicatrix. (Sclerosis.)

Highly vascular adventitious growths are particularly serious, from their liability to swell.

Cancer of the brain very often occurs quite alone in the organism; but medullary cancer, and melanosis, amongst others, are usually associated with cancer in other organs, or with a general development of cancer.

Lastly, there is usually but one adventitious growth in the brain; but when there are two, it is interesting to notice, that they not uncommonly occur symmetrically in corresponding portions of the brain.

g. Entozoa.—The acephalocyst, with its inhabitant, the Echinococcus, and the Cysticercus cellulosæ, are pretty frequently found in the brain. There are often some few only of one or the other, while in some rarer instances they abound in almost countless numbers. In the former case they are usually confined to the brain, in the latter the muscles also are often crowded with them.

They occupy the gray substance almost exclusively, selecting the peripheral much more than the central masses of it. When situated at the surface of the brain, they mostly project beyond its level, and become partially imbedded in the tissue of pia mater.

Should the animal die, the cyst becomes thickened and shrunk, and its contents inspissated, and at last chalky; so that a chalky concretion is afterwards found enclosed in a fibrous bag. It is very difficult to distinguish between this and cretified tubercle, and the diagnosis can be established only by the presence of other living animals.

APPENDIX.

DISEASES OF THE CEREBRAL APPENDAGES. (HYPOPHYSES.)

THE PITUITARY GLAND.

THE pituitary gland is far more frequently the subject of disease than the other hypophysis. Morbid processus, especially those which are deeply seated, have their site in its anterior vascular lobe.

§ 1. *Anomalies in Size.*—Increase in the volume of the pituitary gland may result from congestion, inflammatory swelling, and abscess, or from the presence of an adventitious growth. I have never observed any actual hypertrophy of the gland.

Atrophy, on the contrary, is not unfrequent. It accompanies, in old persons, the same affection of the brain. It prevails especially in the posterior lobe, which, besides diminishing in size, becomes loosened and pulpy (softened), and discolored to rusty brown, or yeast-yellow. At the same time the anterior lobe becomes pale, withered, and tough. Moreover atrophy is sometimes produced, in cases of chronic dropsy of the ventricles, by the pressure which is exerted upon the gland by the serous effusions, through the medium of the third ventricle.

The infundibulum, in like manner, usually shares in the atrophy, and becomes pale, withered, and thin.

§ 2. *Diseases of Texture.*

1. *Hyperæmia.*—Congestion of the pituitary gland is generally combined with the like condition of the pia mater; but in young persons it is occasionally observed alone. It occupies the anterior vascular lobe, and the infundibulum. The cluster of vessels descending on either side of the latter, to the gland, is injected, the substance of the infundibulum is redder than natural, and the turgid gland is loose in its texture, dark red, and full of blood. In some very rare instances the congestion leads to apoplectic extravasation. I have observed small streaks of it associated with congestion of the membranes.

The gland is anæmic in cases of anæmia of the membranes.

2. *Inflammation.*—This process also, so far as I am aware, is confined to the anterior lobe. The appearances which distinguish it are swelling, dark reddening, and looseness of the texture of the gland, but more particularly exudation. This last is usually a yellow, fibrinous product, with small points of which the gland is dotted; or which collects in spots, of the size of millet, or hemp-seed, or runs together in larger masses. Suppuration may take place in these masses, and give rise to abscess of the gland; and this, by the advance of the inflammation in the adjoining parts, may enlarge, and attain a considerable bulk. I have seen cysts of this kind, from the size of a hemp-seed, to that of a hazel-nut. If the inflammation in the adjoining parts terminate in callous induration, the abscess becomes encysted.

3. *Adventitious growths.*—The disease of this class which I have observed in the gland, are tubercle and cancer.

a. Tuberculosis of the gland is, on the whole, a rare disease. It occurs only in combination with tubercle in other organs, especially in the lungs and brain. It assumes both the form of gray crude granulation, and that of a mass of yellow tubercle, which softens and suppurates.

β. Cancerous productions are proportionally more numerous; indeed they are the most common of all the important diseases of the organ. In every instance which I have observed, the growth was of the medullary kind. In one case, in particular, it was a lobulated encephaloid, of a loose, creamy structure, enclosed in a fibrous (neurilemmatous) cyst. In a second case, it was a vascular, reddish-brown, and elastic, but firm medullary growth; while, in a third case, a similar growth was found, degenerated into a brown, chocolate-colored fluid, and contained in a neurilemmatous sac, which projected into the pharynx through an opening it had formed in the base of the skull, by destroying the body and part of the greater wing of the sphenoid bone. In several cases the growth had reached a considerable size, and the base of the skull was destroyed, and the brain displaced upward and laterally, to a corresponding and very marked extent.

The growth in this gland was, in several cases, the only example of the disease in the body; whilst in others, on the contrary, it was associated with cancerous formations in other organs.

γ. In conclusion, there is a morbid growth, belonging to the class of adventitious productions, which consists of a gummy or glutinous (colloid) substance of a yellow color like citron or yellow wine, and occupies the cellular interstice between the two lobes of the gland. If a horizontal section be made, dividing both lobes in half, this growth will be found to form a layer between them, from a quarter of a line to a line in thickness, and sometimes to force the lobes asunder.

The growth in question has acquired importance from the brothers Wenzel having asserted that it was the cause of epilepsy. I have made frequent examinations of the gland, with reference to this assertion; but have as frequently failed to discover the disease in those who had notoriously suffered from epilepsy and convulsions, as I have met with it in other individuals who had been thoroughly healthy.

THE PINEAL GLAND.

The pineal gland is rarely the subject of disease, not only in a general point of view, but even in comparison with the pituitary gland. Its affections are also slight and unimportant. They are confined to—

1. The production of the yellow sand (acervulus) at too early an age (the proper date is fixed by Sömmering at 14 years), and its existence in great quantity, or in large confluent masses; and,
2. Enlargement of the cavity of the gland, and its final conversion into a membranous sac as large as a pea or a hazel-nut, or even still larger. There is usually, in such cases, a large quantity of sand scattered in the wall of the cyst: it is accumulated near the peduncles, where the wall still remains parenchymatous, and it gives the cyst some appearance of being ossified. This change is always associated with a certain degree of chronic hydrocephalus, and with thickening of the lining membrane of the ventricle.

CHAPTER II.

THE SPINAL CORD.

SECT. I.—ANOMALIES AND DISEASES OF THE MEMBRANES OF THE SPINAL CORD.

OF THE DURA MATER OF THE SPINAL CORD.

§ 1. The fibrous investment, and the other membranes of the spinal cord, are but partially developed, or are not developed at all, in cases of acephalus, in which, at the same time, the spinal cord is wanting (amyelia).

§ 2. The principal deviation from the natural size and form of the dura mater is the saccular dilatation met with in partial spina bifida—hydrorachis, of which I shall have to speak hereafter.

§ 3. Its continuity is broken not only by penetrating bodies from without, and by fragments of the vertebræ, when they are fractured and comminuted,—by either of which it may be wounded in various ways,—but also from other causes. Thus we find various openings in it: in cases of extensive spina bifida (atelomyelia of Béclard), it is deficient at its posterior part. It may be lacerated in the cervical region by the odontoid process of the axis, whenever, in consequence of inflammation and suppuration of the ligaments, and of caries, that vertebra is dislocated. Ulcerated openings form in it from the pressure of abscesses, and particularly of those abscesses which are connected with caries of the ribs and vertebræ, &c.

§ 4. *Diseases of its Texture.*

1. *Inflammation.*—Inflammation of the spinal dura mater occurs as a consequence of accidental injuries, and is propagated from adjoining inflamed and suppurating tissues, such as ligaments and bone; but it does not arise from any other cause. Its characters are those of inflammation of fibrous tissue in general; and when it extends more deeply, it gives rise further to circumscribed exudation on the inner free surface of the membrane. If the conditions under which it originated do not destroy life too soon, either by the pressure of the swollen vertebræ on the spinal cord, by spontaneous dislocation of the vertebræ, and laceration of the cord, or otherwise, the inflammation may reach its natural terminations. In those cases in which the inflammation of the bone and the caries heal, the dura mater continues permanently thickened, and adherent to the bone, and even to the visceral layer of the arachnoid also. In other cases, the inflammation of the dura mater terminates in suppuration and perforating ulcer. It may thereupon extend, as a circumscribed inflammation, to the inner membranes, and to the cord itself, or may spread out into a general spinal meningitis.

2. *Adventitious growths.*—They are mainly confined to cancerous growths.

a. Cancer of the fibrous spinal membrane, known as *fungus duræ matris spinalis*, resembles, in its general characters, the fungus of the dura mater of the brain. It may be a primary disease, or the membrane may degenerate secondarily in consequence of being involved in cancer of the vertebræ, and especially of the bodies of those bones. It generally grows exuberantly over a considerable extent of the surface of the membrane, either on its outer or inner side: and as, in the latter case, the arachnoid is destroyed by the advancing growth, the canal of the dura mater is sometimes entirely filled up. Not unfrequently, when it springs from the bones, it involves principally the fibrous sheaths of the nerves and their ganglia in the intervertebral foramina. More rarely it forms circumscribed tumors.

With regard to the form of the disease, it is most commonly the medullary. Its usual seat is the inferior dorsal, and the lumbar regions of the vertebral column.

b. Tuberculosis of the dura mater of the cord occurs in cases of tuberculous caries of the vertebræ, both in the form of an inflammation attended with tubercular exudation, and in that of tuberculous suppuration.

It is remarkable that no production of bone, such as occurs so commonly within the cranium, and is known in that situation as ossification of the dura mater, should ever be met with in the dura mater of the cord; or, at least, it is an extremely rare appearance. Its place is here occupied by the deposition of bone on the free inner layer of the spinal arachnoid.

OF THE SPINAL ARACHNOID MEMBRANE.

The spinal arachnoid, especially in its visceral layer, occupies a different position from that of the cerebral arachnoid, both physiologically and pathologically; for it forms a sac, which does not, as within the skull, adhere closely to the pia mater, but envelopes the spinal cord without being fixed to it. Its position corresponds to that of the cerebral arachnoid, where the latter is stretched across, like a bridge, from one part of the brain to the other. The sac of the spinal arachnoid, therefore, is not single and external only as in the brain, there being also a second space, internal, and enclosed by the visceral layer of the membrane. Generally, however, by the spinal arachnoid sac the inner space is understood. And it is also that which, for its pathological importance, claims, beyond comparison, the most attention; the visceral layer, and the sac within it, being the seat of the most serious diseases of both the inner membranes of the cord, as well as of the products which those diseases furnish.

§ 1. *Anomalies in the Extent or Dimensions of the Membrane.*—Besides the uniform enlargements to which the (inner) arachnoid sac is subject from exudations and dropsical effusion into it at various periods of both intra- and extra-uterine life, further anomalies are met with, belonging to the same class;—congenital partial dilatations, which, when combined with local (partial) fission of the vertebral column, represent the disease named Spina bifida. The arachnoid protrudes through an opening in the arches of the vertebræ,—generally an opening left in the mesial line by the deficiency of some lumbar or sacral spinous processes,—and forms a sac or diverticulum, which is filled with serum, and the cavity of which communicates through the neck of the sac with the chamber that contains the cerebro-spinal fluid. The dura mater of the cord, extremely attenuated, is spread over it; but more commonly the arachnoid protrudes through a round or elongated opening in the dura mater also, and is then covered immediately by the general integuments. The sac varies in size, from a scarcely perceptible dilatation to the bulk of a duck's or goose's egg, or it may even be larger. The fulness of the sac, and its consequent tension and fluctuation, are much diminished after death, when it is more or less collapsed. Its base is always constricted, even when broad; and it sometimes forms a pedicle.

The sac is composed of arachnoid membrane, and of general integuments, which are more or less attenuated in proportion to its size, and marked with scars, when it has previously burst. The canal by which its cavity communicates with the internal arachnoid space is short, and always narrow when compared with the area of the protruded sac, and it corresponds with the aperture in the posterior wall of the spinal column, and in the dura mater.

The spinal cord itself may be fissured; more or less of it may be wanting; or it may be quite natural. The nerves may present no further anomaly than that of being stretched longitudinally, but they usually separate from the spinal cord, and, gradually becoming thinner, and wasting, they terminate in the arachnoid, at the base of the sac. This is very commonly the case in somewhat large sacs in the lumbar and sacral regions, in which the dilatation of the arachnoid cavity is aided by a further and important agent in the separation of the nerves, viz., the receding of the spinal cord upward in the latter period of fœtal life.

The direct consequences of this vary with the circumstances of the case; there may be slight (Paresen), or complete palsies, which are generally combined with tonic cramp and contraction (club-foot, &c.); or there may be insufficient nutrition. When the tumor is compressed, general convulsions come on, and if the pressure be increased, coma ensues. The general integuments are sometimes so stretched that they inflame and slough, and, in this condition, they rend wide open. The latter accident sometimes occurs during birth, and the sudden evacuation of the contents of the sac is frequently rapidly fatal.

When the nerves and spinal cord are not involved in the disease, it is capable of cure, like the dropsical sacs of the cerebral arachnoid, by being gradually emptied, or by ligature. It may be cured spontaneously by a single, or by repeated opening of the sac, and even by spontaneous separation of the sac, and obliteration of its neck within the fissure in the vertebræ.

In a few cases spina bifida continues to exist up to the period of puberty, and even beyond it: it then not unfrequently increases in size.

It is very commonly, if not always combined with hydrocephalus, and thereby with hydrencephalocele, and hemicephalus.

The origin of spina bifida is traceable to the same cause which gives rise to dropsical sacs of the cranial arachnoid. A congenital dropsy of the arachnoid sac sometimes occurs, which is closely allied to spina bifida, and in which, whilst the sac is uniformly dilated, the vertebral canal maintains its integrity.

§ 2. *Diseases of its Texture.*

1. *Hyperæmia. Apoplexy. Inflammation* (*arachnitis spinalis*).—No congestion of the spinal arachnoid occurs, or inflammation of it to any extent, without a similar condition of the pia mater of the cord: and, as the vascularity is much more marked on the latter membrane, the description of these affections and of their products may be postponed till the diseases of the pia mater are taken into consideration.

There is, however, one condition,—a consequence of habitual, long-continued, or repeated congestions, and of slight attacks of inflammation, which, as it is an affection of the arachnoid membrane almost alone, demands notice in this place. It consists of dulness, opacity, and thickening of the arachnoid, and is usually combined with chronic effusions of serum into its sac (mostly the inner sac). These changes are sometimes diffused uniformly over a certain portion of the membrane; at other times they are developed at separate spots, and form insulated patches (plaques): but very slight indications, if any, ever occur of a granular thickening, corresponding to the Pacchionian bodies observed in the cerebral arachnoid. The lumbar portion of the membrane is the most affected; and there, indeed, habitual congestions most frequently occur. The so-called ossification of the spinal arachnoid has some connection with these congestions.

Adhesions between the visceral layer and the dura mater rarely take place, except as the result of local, and especially of traumatic, inflammations of the arachnoid.

2. *Adventitious growths.*—All forms of growth are very rare in this membrane; except *bone*, which forms a marked exception to the rule.

It is always found on the visceral layer of the membrane, and forms scales and plates, which are generally of small size; ranging between that of a poppy-seed and that of a lentil, and but rarely equalling a pea or a silver threepenny-piece (Groschen). They are cartilaginous, or cartilaginous and bony, flexible, and of a white, or yellowish-white color. They are bent towards the periphery of the cylinder, and appear convexo-concave; and the larger they are, the more distinct is this peculiar form. They are imbedded in the arachnoid, but they project from the membrane inward rather than outward; their inner surface is rough, the outer smooth. Their number is very various; sometimes at one, or a few scarcely perceptible points only, the commencement of the bony growth can be detected; in other cases they are very numerous. Their usual situation is the lumbar and lower dorsal regions; but, in a few instances, when they are very numerous, they may extend as high as the upper dorsal region.

Ollivier speaks also of *cancerous* tumors almost free in the arachnoid sac.

§ 3. *Anomalous Contents of the Spinal Arachnoid.*—As these are principally products of the vascular system of the pia mater, I shall treat of them among the diseases of that membrane. For the same reason, the morbid effusions are almost always confined to the inner arachnoid sac.

OF THE PIA MATER OF THE SPINAL CORD.

§ 1. *Diseases of Texture.*

1. *Congestion, Apoplexy.*—The anatomical characters of this congestion are too evident to require description. The subjects of it are mostly those in the tenderest childhood; infants just born or suckling, and individuals in the years of growth. In the former, the congestion generally involves the pia mater of the whole spinal cord, and that of the brain also; in the latter, on the contrary, it is generally habitual, and confined

to the lumbar region of the cord, and is combined with distension of the sinuses in the vertebral canal, and with congestion—so-called hemorrhoidal congestion—of all the pelvic organs.

When the congestion is excessive, it becomes fatal as vascular apoplexy; but sometimes that termination is anticipated by the occurrence of hemorrhage into the sac of the arachnoid (apoplexia gravis). A quantity of blood, most of it loosely clotted, is then found filling the sac, sometimes even up to the medulla oblongata. This is observed especially in the individuals first named.

Amongst the results of spinal congestion, acute and chronic effusions of serum into the arachnoid sac may be noticed. The former, the acute, constitute serous spinal apoplexy, which also occurs mostly in children. The serum is generally of a yellowish color, or yellowish tinged with red, and after it is effused, the vessels still remain overfilled. The chronic effusion consists of colorless clear serum. Opacity, thickening, or ossification of the arachnoid coexist with them, and dilatation of the sac in the lumbar region: congestion is not necessarily present with the chronic effusion, though there is abundant evidence in the dilatation and varicose state of the vessels of the pia mater, and in the condition of the arachnoid before mentioned, of its previous repeated occurrence.

The chronic effusions so common in advanced age, are, in part at least, consequences of the congestions brought on by atrophy of the medulla and nerves.

2. *Inflammation* (*meningitis spinalis*).—Inflammation of the pia mater of the spinal cord succeeds various internal injuries; and it may pass over to the membrane from adjoining inflamed tissues: but, besides being thus set up, it sometimes, though very rarely in comparison with cerebral meningitis, comes on spontaneously. It is very often combined with cerebral meningitis, and occupies the whole spinal cord; and it is a disease particularly frequent in early childhood (Billard). The characters of the inflammation are the same as those of cerebral meningitis, but there are several peculiarities about the exudation.

The greatest part, and even the whole, of the product of the inflammation may be poured out upon the surface of the pia mater, into the inner sac of the arachnoid: one layer of it then hangs loosely on the cord, enveloping especially its posterior part; while another and still larger portion, mixed with more or less serum, is accumulated in the arachnoid sac, especially behind the medulla. The pia mater is rendered slightly turbid, and is swollen by the serous portion of the exudation, but its tissue contains very little plastic product, and that little but rarely. The cord generally appears pale and anæmic, and occasionally its texture is somewhat loosened and softened by symptomatic œdema (serous infiltration). The explanation of these appearances is found, on the one hand, in the peculiar relation which I remarked at the commencement, as subsisting between the spinal arachnoid and pia mater; and, on the other, in the density of the structure of the pia mater, and the closeness with which it adheres to the medulla.

The exudation is usually of a pure yellow, or greenish-yellow color, coagulable, fibrinous, or purulent. I have never had occasion to suspect it to be of tuberculous nature: and this observation accords with the facts, that spontaneous spinal meningitis so commonly coexists with that form of cerebral meningitis which produces similar exudations, and that tubercle of every kind is extremely rare in the pia mater of the cord. An acute tuberculosis, indeed, so far as I am aware, never takes place in it.

3. *Adventitious growths.*—They are all, as I have remarked about tubercle, extremely rare. Ollivier mentions a case of encephaloid, which formed a layer, adhering to the posterior part of the pia mater, from one end of the cord to the other.

SECTION II.—OF THE SPINAL CORD.

§ 1. *Deficiency and Excess of Development.*—A total absence of the spinal cord, such as occurs when both the head and the trunk are wanting, is rarely met with: but a partial deficiency of it is more frequently observed. In cases of acephalus the defect is proportioned to the extent to which the neck, thorax, and even the abdomen are wanting. Allied to this is another partial deficiency, in which parts of the cord are arrested in their development in bulk: thus, when the upper or lower extremities are wanting, or withered, there is no enlargement in the cervical or lumbar regions of the cord.

An original deficiency, whether complete or partial, may be distinguished from that which, being produced by the destruction of the fœtal medulla, merely has the semblance of being original, in just the same manner as hemicephalus and acephalus are distinguished from each other (p. 280). Hydrorachis is, I believe, as much known by the name of spina bifida, when it is a disease of the spinal cord, as when, in the form of fœtal dropsy, it affects only the spinal arachnoid sac (p. 252),—provided only the vertebral column be fissured. And I believe it will be best to treat of that disease in this place, referring to what has been said at p. 176, for the condition of the vertebral column.

In its highest degree, the cord is altogether wanting, and the nerves terminate on the inner side of the arachnoid. The vertebral canal is then usually wide open posteriorly—fissured, that is, to a great extent; the spinal membranes, especially the dura mater and arachnoid, are intimately united with one another, and shreds of pia mater hang within the latter from the extremities of the nerves. The membranes, like the bony canal, are open posteriorly; and the general integuments, deficient to the same extent, adhere to them on both sides at the margin of the fissure. Not unfrequently a streak of hair extends along the margin of the skin from the head downward, and just overhangs the line of adhesion. The posterior surfaces of the bodies of the vertebra are thus laid bare, being covered merely by the spinal membranes.

Sometimes, as an exception to this arrangement, the membranes of the cord are not open, but form a closed bag filled with serum. In some very rare cases of this kind the vertebral column also is not fissured.

In a less advanced degree of the disease, rudiments of the cord remain, and especially its anterior columns, more or less complete.

In a still less degree, the spinal cord is fissured, and appears open behind; it is broad and flat. The membranes and the vertebral canal are in the condition mentioned above.

Allied to this is an extremely rare case, in which the canal of the spinal cord still remains, and is dilated in consequence of chronic dropsy within it.

The degrees just spoken of are measured by the amount of destruction of the cord, and by the arrest in its complete development, without reference to their local extent. Each degree may affect the whole length of the cord, or only portions of it; and the simultaneous fissure in the vertebral column may constitute accordingly either a total or a partial (local) spina bifida. The latter may exist at any portion of the spine, or at two different parts in the same subject; the spinal membranes are then either fissured in the manner above described, or they are merely distended, and form the well-known membranous sacs in the spinal region described at page 252.

Every case of hydrorachis is, usually at least, combined with hydrocephalus; but when the whole spine, or its cervical region, is fissured, the kindred disease, hemicephalus, or else hydrencephalocele, is always present too.

Hydrorachis is similar in its nature to congenital hydrocephalus; and when it reaches advanced degrees, it produces spina bifida, just as hydrocephalus occasions hemicephalus and hydrencephalocele.

The consequences of hydrorachis, are self-evident, when reference is made to what has been already said on the subject of dropsical arachnoid sacs.

Spina bifida in its slightest degree is occasioned by dropsy of the arachnoid sac; the cord maintains its integrity, but the development and closure of the arches of the vertebræ are arrested, and the arachnoid bulges through the apertures in the posterior wall of the vertebral canal: it is generally only partially dilated, and forms a sac, such as has been already described (p. 252). The chief situation of this form of spina bifida is the lumbar, lower dorsal, and sacral regions.

In a few cases the cord itself passes through the fissure in the vertebræ, and a hernia of the spinal cord takes place.

Excessive development of the spinal marrow is met with only in double monsters. Its form varies according to the extent of duplication, &c.

§ 2. *Anomalies in Size.*—The length of the spinal cord generally has some relation to that of the vertebral column: so that, for instance, in individuals in whom a tail exists, the cord is unusually long. But sometimes there is more or less of disproportion between the two: the cord is unusually short; or being unusually long, it suggests the recollection of its condition in the fœtus. Thus, for example, in fission of the vertebræ (spina bifida) it is in part of really unnatural length, and partly it has the appearance of being too long, because the vertebræ are arrested in their development, and frequently at the same time malformed.

The thickness of the spinal cord is subject to many peculiarities in different individuals, as well as to various local anomalies, which affect particularly its enlarged portions, and depend on deficient or excessive development at the periphery of the nervous system.

Considerable morbid enlargements in the bulk or diameter of the cord result from various anomalies. Some of the enlargements are local, and are bounded by a more or less sharp margin; while others, on the contrary, extend throughout the whole cord. The principal are congestion, apoplexy, œdema, inflammation, serous effusions into a persistent fœtal canal, similar effusions which fill up the space when the gray tract is atrophied, morbid growths, and so on, and lastly hypertrophy. The last is the only one which properly belongs to the present section.

Hypertrophy of the spinal marrow is exceedingly rare when compared with that of the brain. Although some of the many cases which have been adduced as hypertrophy are certainly of a different nature, and others appear to be at least doubtful, yet there can be no doubt that such a disease really does occur. It affects in some instances the whole cord, and is then almost always combined with hypertrophy of the brain, which forms the more prominent feature in the case: in other instances it is partial, and especially affects the enlargements of the cord; or at least they appear to be its original seat, and from them the disease extends to the other parts of the cord.

Ollivier holds the swelling which the marrow presents near a part that has been subject to pressure, especially above such a part, to be hypertrophy. If this swelling be not produced by some distension of the medullary tubes, by an enlargement resulting from the displacement of their contents out of the compressed part, yet it may resemble the secondary form of hypertrophy of the brain, that, for example, which arises from the irritation of growths in the brain; or perhaps, more commonly still, it may be a condition in which congestive swelling, œdema, and hypertrophy all meet together. It reminds us of the hypertrophy which the brain undergoes when the medulla oblongata is subjected to pressure (p. 285).

The character of the spinal marrow, when hypertrophied, is, on the whole, analogous to that of the hypertrophied brain: its form becomes round and plump, and its grooves seem smoothed out; it has a peculiar turgescence, while at the same time the cord and the pia mater are anæmic; its consistence appears to be firm, but when the membranes are removed and the cord itself is pulled asunder, it is found to have the consistence of dough.

It occurs mostly in children, and sometimes even in the fœtus. Ollivier has noticed it in several cases affecting the gray substance.

A more frequent and a better-marked affection of the cord is atrophy, and diminution of volume to which it gives rise. Atrophy occupies either the whole cord, or merely separate portions of it. Most frequently total atrophy is but partial at first, and gradually extends till it occupies the whole length of the cord.

Total atrophy of the spinal marrow comes on in old age simultaneously with the same affection of the brain (atrophia medullæ senilis). Though it is equally a primary disease in the medulla as in the brain, yet it is usually better marked in the latter organ. Its characteristics are identical with those of senile atrophy of the brain, diminution of volume, and dirty whiteness of the fibrous columns, a rusty-brownish, fawn tint of the gray substance, and toughness of the white substance, even to a degree like that of leather. Chronic effusions of serum in the sac of the arachnoid accompany it, and opacity, thickening, and ossification of that membrane. Not unfrequently the cord, discolored as before mentioned, is infiltrated with serum, and its substance is then soft and withered. Like the corresponding atrophy of the brain, it may come on as a premature marasmus.

The partial atrophies are, most probably, in many cases consecutive upon disease at the periphery of the nervous system. They affect particularly the enlargements of the cord. Atrophy of the lumbar portion especially, under the names of Tabes dorsalis, has long obtained the attention of physicians, as a consequence of decay of generative powers, and spermatorrhœa. From thence the wasting gradually extends, as has been said, and becomes a total atrophy.

These atrophies attain various degrees, which are determined simply by the diminution in the volume of the cord. They vary so much, that in one case the wasting is but just discernible, while, in another, the cord may have but half, or scarcely half, its natural thickness. There are differences, also, in other respects: thus the white columns vary in their consistence, sometimes being natural, but more frequently compact, dry, and even as tough as leather.

In some few cases of considerable wasting, the cord is found constricted here and there, and nodose, a series of bulgings being produced by successive constrictions of the cord.

From numerous observations I may assert, generally, that the more advanced degrees of atrophy, such even as are palpable at once, and not those which are discernible only upon comparison with other preparations, are rare.[1]

The secondary atrophy which the cord undergoes, in consequence of diseases of its texture, especially of inflammation, will be described below.

Lastly, when the cord is subjected, in any part, to the pressure of enlarged, or of dislocated (inflamed and carious) vertebræ, of exostoses, aneurisms, morbid growths, &c., it becomes flattened and thin, in proportion to the amount of pressure. In such cases it is found swollen and thickened, especially above the compressed spot, and, very commonly, below it also. This condition, as I have before remarked, is distinctly asserted by Ollivier to be one of Hypertrophy.

§ 3. *Interruptions of Continuity.*—Various instruments penetrate to the cord, and inflict upon it incised, punctured, or shot-wounds. Much more frequently it is contused and lacerated by fractured, crushed, comminuted, or dislocated vertebræ: but sometimes these injuries are occasioned merely by excessive stretching and concussion of the spinal column. The most remarkable and important of the injuries which the cord undergoes in consequence of luxation of the vertebræ, is that in which sudden death ensues, immediately upon its being crushed or lacerated; such an event occurs when those bones are dislocated spontaneously, and especially when, in consequence of inflammation and suppuration of the ligamentous apparatus, and of caries, the odontoid process of the second cervical vertebra becomes loose, tears through the dura mater, and is dislocated into the vertebral canal.

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[1] [The construction of this sentence seems to indicate that a word has been omitted, and that the author would say, such degrees of atrophy are *not* rare.—ED.]

Sometimes only a part of the cord is bruised: at other times it is changed in its whole thickness, and more or less of its length, into a suffused red pulp; or it is crushed quite through. In the last case, the two ends of the cord are held together by the pia mater; but if that also be torn, they remain opposite to each other, and quite free. The inflammation and yellow softening of the cord, which result from injuries of this kind, usually carry them on to a fatal termination; but sometimes it is by the paralysis and its consequences that life is destroyed.

§ 4. *Diseases of Texture.—Congestion, Apoplexy.*—Congestion of the spinal cord is a symptom in the course, and a sequel, of sundry acute and chronic diseases. Such, most probably, is its real import, in those who have died of tetanus, convulsions, and hydrophobia. It is almost constantly met with in those cases, combined with an equal degree of congestion of the brain. In other cases, it is set up by inflammatory processes in the vertebræ, by the pressure and irritation of exostoses and swollen or luxated vertebræ, and by morbid growths in the cord itself: and it leads to permanent turgescence and hypertrophy of the medulla.

But it also comes on idiopathically. It then usually pervades the whole cord and its membranes, as well as the brain, and is most frequently met with in early childhood: in adults it is confined to the lumbar portion of the cord. (Compare p. 330.)

I have had occasion to attribute several affections to what is called spinal irritation; and subsequent communication with the physicians who had had the treatment of the cases, has proved that I have done so correctly. Sometimes there existed a certain amount of congestion, but very often there was neither congestion nor any other unnatural appearance.

Spinal apoplexy, that is to say, hemorrhage independent of injury, is one of the rarest appearance. When it does occur, it is at the cervical portion of the cord, and almost always in the gray substance.

2. *Œdema.*—In both its acute and chronic form œdema frequently affects the cord, infiltering, loosening, and producing what is called "white softening" of its texture. What has been said in general of œdema of the brain, applies also to that of the cord. When it extends over the whole cord, the brain is usually in the same condition.

3. *Inflammation.*—All that has been said of inflammation and red softening of the brain, applies in every respect to inflammation of the spinal cord: only the latter is in comparison much less frequent. In the cord, as in the brain, inflammation attacks sometimes the white tracts, sometimes the gray substance, sometimes both together. But it is a striking fact, and worthy of especial notice, that inflammation of the gray substance affects that part in long streaks, or in its whole extent, restricting itself to that substance, and producing a peculiar condition of the cord as well by the increase of volume which attends its softening, as by a peculiar form of dropsy in which it occasionally terminates. The red softening of the gray substance which accompanies it, and which is tinted according to circumstances, of a chocolate-brown, or a plum-sauce color, rusty brown, or yeast-yellow, corresponds with the *central softening of the spinal cord*, described by Albers.

The following three cases will be found to represent the characteristics f this process; the preparations taken from them are in the Vienna Museum.

Case I.—P. Joseph, æt. 23, a journeyman joiner, was admitted into he General Hospital, January 19, 1835. He stated that three weeks efore admission, he had stood a considerable time in cold water, and ad in consequence experienced violent acute pain in the feet and back, ccompanied with fever. A few days afterward, tremulous motions and oss of sensation came on in the lower extremities. On his admission he was unable to stand, though he could move his feet in bed; and he had difficulty in passing his urine: he had no fever. In another fortnight he power of moving the legs was quite lost, the urine accumulated in he bladder, and produced a burning pain as it was passed: the vesical region became painful: fever supervened, and frequent vomiting. In March a slough formed on the sacrum, bloody urine was passed through he catheter, and on the 29th of the same month, the patient died.

Examination of the body.—There was a large slough on the sacrum. The penis and scrotum were swollen, and on the latter were several dirty brownish sloughs.

The lungs generally were œdematous, and their upper lobes tubercuous. The kidneys were large, their pelves were somewhat dilated, the urinary bladder was moderately distended, and its mucous membrane was covered with an exudation which was a line in thickness; whilst underneath it was of a blood-red color, injected, and here and there changed into a brown slough. The cellular tissue of the scrotum was illed with a brownish fetid ichorous fluid.

Spinal cord.—There was a small quantity of serum in the arachnoid ac. The spinal cord was swollen in the lower part of the neck, and till more so in the upper dorsal region; in the latter part its gray subtance was infiltered with a grayish-yellow exudation, and was itself in a state of red and yellow softening (gelbröthlich erweicht). About the econd and third dorsal vertebræ, the place of the gray matter was occupied by a firmly fixed cylindrical plug of yellow exudation, more than an inch in length, and from three to four lines in thickness.

Case II.—St. Leopold, æt. 22, after a fall on his head, three years ago, had been suffering from pain, which was at first slight and transient, and confined to the occiput, but subsequently became severe and continued, and affected the whole head; it at last extended to the nape of he neck, and the upper extremities.

In the second year of the disease, after a feverish attack, which lasted fourteen days, his spinal column began to bend, and he became subject o continued vertigo. All the symptoms diminished while he was in the ecumbent posture, and increased after taking a meal: the latter, moreover, was always followed by ineffectual efforts at vomiting. On his admission into the hospital, he had, on the whole, a florid appearance: the symptoms above mentioned were all present; the countenance was ruddy; the head hot; the pulse was accelerated; the respiration natural; and he bowels at times were sluggish. During two months in which treatment was employed, the symptoms at times abated, and then grew worse again. In the latter part of his life, erysipelas came on in the face: for the last two days he had pain in the bowels; and some hours before his death, he lost consciousness, and had paralysis of the lower extremities.

Examination of the body.—The body was pretty well nourished. The pia mater was infiltrated, and the surface of the brain pale: its white substance was also pale, it was moderately vascular and soft, and contained serum. The vessels at the base of the brain, especially about the pons, were dilated and tortuous.

The lungs were of a dark-red color, and congested; the upper lobe of the left was œdematous.

In the peritoneum there was a pound of serum mixed with flakes of lymph. Numerous hemorrhagic erosions, as large as pins' heads, dotted the mucous membrane of the stomach. The liver was shrunken, and of a deep-yellow color; a few drops of dirty grayish-yellow bile were found in the gall-bladder. The follicles of the small intestines were swollen. The colon contained grayish clay-like fæces. The kidneys were flabby and vascular: the urinary bladder was contracted and contained half an ounce of turbid urine.

The vertebral column curved in its upper dorsal region backward, and to the right. The vertebral venous sinuses were very full of blood; the dura mater was relaxed; the arachnoid was opaque, and in the lumbar region contained several small plates of bone. The spinal cord was swollen, and felt soft and fluctuating. The white columns were distended into a kind of bag, and enclosed instead of cineritious substance, a grayish briny fluid infiltrated through a loose delicate cellular structure. The change extended upward nearly to the medulla oblongata and downward beyond the lumbar enlargement of the cord.

CASE III.—L. Katharina, æt. 40, eighteen years before death, had a fall from the top floor of a house, by which the spinal column was fractured in the lower dorsal region. The injury got well with an angular curvature in the back, but left behind it an unsteady, dragging gait, and incontinence of urine. While she was in the hospital, the most marked symptoms were redness of the general integuments, and pain on moving. She died with fever and diarrhœa, extremely emaciated.

Examination of the body.—Much emaciation. The left lower extremity was œdematous. The vertebral column was bent at an obtuse angle in the lower dorsal region, and above that point was slightly curved in the form of the letter S.

There were cellular adhesions of both lungs, and parietal œdema, and lobular hepatization of the right lung posteriorly. In the right upper lobe there was an abscess as large as a hen's egg, and a second as large as a hazel-nut, close to the pleura.

(The gall-bladder was distended with a fluid, like white of egg; and its neck was obstructed by a crystalline stone as large as a dove's egg.

The mucous membrane of the large intestines was strewn with stalked granulations.)

The kidneys were large. The urinary bladder was contracted and empty; and spots of its mucous membrane were softened, dark red, and injected. The right ovarium was converted into a sac of fat larger than a hen's egg.

The substance of the brain was pale and tough.

The dura mater at the base of the skull, especially at its basilar part, and in the neighborhood of the sella turcica, was of a dark-red color, and covered, especially in the former situation, with a yellowish exudation. The cavernous sinuses, the circular sinus, the anterior occipital, and the commencement of the petrous sinuses, were filled partly with a brown friable coagulum of blood, but for the most part with a brown and yellow purulent fluid. The left crural vein, and its branches down to the leg, contained a brown coagulum, the outer layers of which were softening, and becoming purulent; whilst within the pelvis it was filled with a brown ichor-like fluid. The commencement of the hypogastric veins, and the iliac, up to the cava, were filled with a similar fluid. The coats of the veins were thickened and discolored, the inner coat being loose like nap, and dull.

At the part where the column was curved, the canal through the vertebræ was contracted to a narrow fissure. A good deal of serum was accumulated in the sac of the spinal arachnoid, and, in the lumbar region, that membrane was occupied by small osseous plates, of about the size of lentils. The pia mater was traversed by distended vessels, and in the lumbar region, infiltrated with a briny fluid. The spinal cord, from the dorsal curvature to the second cervical vertebra, was converted into a fluctuating bag, which was composed of the white columns, and contained a clear serous fluid, but no gray substance, that having disappeared. The columns were softened and pale: the posterior middle columns were forced asunder more than half a line. The inner wall of the bladder was lined by a delicate cellulo-serous tissue, which projected inward, forming numerous ledges, and also extended in bands across the canal from one wall to the other.

Viewing these cases in order, we observe in the first a very intense inflammatory process (red softening) in the gray matter of the cord, distinguished for the quantity, and plastic nature of its products; the gray matter was augmented in bulk, whilst the white columns around were distended. In the second case, the absorption of one part of the products had commenced, and of the substratum, in which the process was going on, while another part of the products was in course of conversion into a cellular structure, and was infiltered with serum. In the third we find the issue of the process: in the place of gray substance there remained a cavity lined by a part of the products of the inflammatory process organized to a tissue, and constituting the cellular infiltration of Durand-Fardel (p. 310). We have here a dropsy of the spinal marrow, quite peculiar, both as to its cause, and as to its nature: the cases in which it has occurred have been recently collected and arranged by Nonat.

In consequence of inflammation (softening) involving the whole thickness of the medulla, the cord has been found completely divided.

4. *Softening and induration.*—The same forms of softening which happen in the brain, are found to occur in the spinal marrow also. But, on the whole, they are more rare in the latter. *White* softening very seldom reaches that degree of complete destruction of the nervous tissue, which is so frequently met with in the brain, especially in the neighborhood of the ventricles. *Red* softening (myelitis) assumes the very remarkable form which I have just described as central softening of the cord.

Yellow softening may, without doubt, occur in the cord as a primary and idiopathic disease; but my own observation has furnished me with instances of its secondary form only in and around spots of inflammation, and encompassing morbid growths.

Induration of the cord, though comparatively rare, yet does occur in all the forms, and with all the import which were asserted of it in the brain. Indurations of slight degree are the sequels of general diseases, and are unaccompanied by any manifest further anomaly. More considerable indurations are combined with atrophy. And lastly, condensations and callosities of parts of the cord, are met with as consequences of inflammation, apoplexy, and morbid growths. They are seated mostly in the white columns.

5. *Morbid growths.*—Growths of this nature, even those which, like tubercle and cancer, are frequent in the brain, are very unfrequent in the spinal cord.

a. Tubercle I have observed only in combination with other advanced tuberculoses. Its principal seat is the cervical or lumbar part of the cord, where it sometimes occupies the white fibres, sometimes the gray substance. As in the brain, it leads to inflammation (red softening) and to yellow softening. I have never seen a tuberculous cavity in the cord. Sometimes several tubercles are grouped together, none exceeding the size of millet- or hemp-seed; at other times only one exists, which is of large dimensions equalling a pea or a bean.

b. Exclusively of several cases of circumscribed callous induration of the white columns, as to the cancerous nature of which I am still in doubt, I have met with but one case of *cancer* of the cord. It was a solitary nodule of medullary cancer. Ollivier mentions several examples of diffused carcinomatous growths, as well as of so-called colloid cancer.

c. Among the entozoa, I have repeatedly seen the cysticercus in the cervical portion of the spinal marrow. The acephalocyst sacs, so far as has been observed, have no connection with the cord, their nidus is even outside the dura mater. In one case the cyst forced its way into the canal of the arachnoid.

CHAPTER III.

ANOMALIES AND DISEASES OF THE NERVES.

I VENTURE to treat of all these under one head, introducing the affections of the ganglia at the end of each chapter, somewhat in the form of an Appendix. To subdivide diseases of the nerves into such as affect the neurilemma, and such as involve the fibres, in the same manner as the affections of the central organs were divided into those of the organs themselves, and those of its membranous investments, is for the most part impossible: for, on the one hand, those elements are in the nerves too intimately united, and on the other, our information on the subject is too defective to permit such a distinction. And not only is it impossible, but if the necessary attention were paid to the subject, the distinction would prove of no real value.

§ 1. *Deficiency and Excess of Development.*—The nerves seem never to be altogether wanting, even in those monsters which are most imperfect; but a single one, or even several, are not unfrequently so. Not only are no nerves developed when the parts which they should supply do not exist, but they are defective, though not perhaps quite absent, when portions of the body are incompletely formed; as when there are limbs, but no muscles within them. The most frequent instances of deficiency are those of the olfactory and optic nerves, when the nose, the eyes, or the whole face are wanting, or arrested in their development. Usually, when no eyes have been formed, or when their development has been arrested, the third, fourth, and sixth pair, and the first branch of the fifth are deficient too. In a cyclops in which the face was almost entirely wanting, no facial nerve could be found; nor can even the lingual branch of the fifth pair, or the hypoglossal, when no tongue is developed. (Tiedemann.) "In monsters born without extremities, or with only imperfect ones, the deficiency of the limbs corresponds exactly with that of the nerves; for when single fingers or toes, a hand or foot, a forearm or leg are wanting, their nerves are wanting too; if there be a deficiency to the extent of a whole limb, the brachial or the lumbo-sacral plexus is small, appears entangled, and gives no branches except to the neighboring part of the trunk. If, with the arm, the shoulder should be absent, or half the pelvis with the lower extremity, their nervous plexuses are absent also, and only some posterior branches for the muscles of the neck, or for the lower part of the dorsal muscles, pass out through the intervertebral foramina. In those monsters, not unfrequently found in the human species, which are without the radius and thumb, the radial nerve ends at the elbow, or merely sends down a few slender threads to the back of the hand." (Otto.)

Lastly, there are frequently no nerves in supernumerary parts, especially such as are imperfectly formed.

The absence of a nerve is often only apparent; when the periphery is examined, as it always should be to determine the deficiency absolutely, the nerve will sometimes be found running in one sheath with some other nerve. A nerve sometimes appears to be wanting, when it is only partially so, the trunk of the nerve not reaching the central organ.

Excess of development is illustrated in a plurality of the nerves. An unusual division of a nervous trunk into its branches, or a division of it in an unusually high position, presents merely the appearance of plurality. A real plurality occurs only when there is an increase in the number of other corresponding parts of the body; for instance, there is one spinal nerve more when there is an additional vertebra, and more digital nerves when there is a perfectly organized supernumerary finger. In double monsters, the nerves are doubled and divided according to the manner and degree of the duplication; or they are double, but communicate in parts of their course; or they are originally double, then unite, and remain single.

As it has been noticed that the absence of nerves is often only apparent, so may it also be observed that an excess of development is sometimes only apparent too. Sometimes the unusual branching of a nervous trunk gives it the appearance of being developed to an unnatural degree.

Deficiency and excess in the development of the ganglia are exemplified, on the one hand, in their absence or unusual smallness; and, on the other hand, in their presence on nerves where they are not generally found, or in their existence in unusual number or size.

§ 2. *Malformations; and anomalies in the origin, course, and branching of nerves.*—Anomalies in the form of the trunk of a nerve are very seldom of any importance; instances of it are met with in the olfactory nerves, which are sometimes hollow at birth; and in the optic, which are found in cyclops monsters to be single, or in various extent fused together.

Malformations of the brain occasion many variations in the origin and course of the nerves, even in individuals who are otherwise naturally formed. The best examples of this are afforded, in cyclopia, by the optic and four following nerves of the eye; by the third nerve when it gives off the long root of the ophthalmic ganglion, and by the sixth, which may supply a nasal branch that is lacking from the fifth. (Otto.)

Next to these may be arranged anomalies in the branching of nerves, and in the formation of anastomoses and plexuses, unusually high divisions, splitting of a nervous trunk in the form of islands, deficient, multiplied, uncommon anastomoses, &c.

§ 3. *Anomalies in Size.*—These anomalies relate to the thickness of the nervous cords. A palpable increase of all the nerves beyond their natural bulk is extremely rare; a few instances of it only having been observed in idiots, either affecting all the nerves uniformly, or predominating in the sympathetic system. It is more frequently met with in a single nervous trunk.

It has various causes, but the most common are inflammation, inflammatory products, and morbid growths. A more important question is that relating to the increase in size which nerves acquire by hypertrophy.

There is no proof that hypertrophy of a nerve consists in the formation of new fibres in addition to those of which it was originally composed, and the present state of our knowledge opposes any such explanation. It might be, indeed, that the nerve-tubes become enlarged by an increase of their contents; but this, also, is a mere hypothesis, which, although in some degree supported by the enlargement of the tubes, which Herrmann Nasse has observed at the proximal end of a divided nerve before it is completely restored, can yet receive no practical proof of its correctness, even from a future generation. Most probably the neurilemma of the nerve is the part which is hypertrophied. Such we must, for the present, consider to be the state of the enlarged nerves of hypertrophied organs, those, for instance, of a hypertrophied heart.

There might, however, be a true hypertrophy of the ganglia, an aggregrating of new ganglion cells; although of it also we lack proof from facts. I have met with a case of general emaciation, combined with an eminent degree of hypochondriasis, in which the central abdominal ganglia were considerably enlarged.

It is a peculiarity frequent in individual conformation, that the nerves are more or less manifestly thin and slender. Very commonly when the brain is hypertrophied, there is a striking thinness of the cerebral nerves. The nerves, too, of parts which are developed but imperfectly, or not at all, are thin from the first: an instance of this is met with in the optic nerves in cases of microphthalmus.

The nature of atrophy is more distinct than that of hypertrophy. Sometimes it is confined to the nerve-tubes, sometimes it involves the neurilemma also. There are various conditions under which it occurs.

1. It is only when general emaciation has advanced to an extreme degree that genuine atrophy of the nerves can be shown to exist with it. In ordinary cases the diminution in their size may be referable to the loss of their natural moisture, and, after that, to wasting of neurilemma.

2. Pressure and stretching often produce atrophy of a nerve, even to such an extent as gradually to destroy it in its whole thickness. We see force of this kind exerted upon nerves, morbid growths, aneurisms, and enlarged organs: especially we might instance the bronchial plexuses of the vagus nerve, which sometimes become so wasted from the presence of tuberculous bronchial glands, that their continuity is at last destroyed. The nerve is at first not unfrequently flattened, its fasciculi, spread abroad, and at length it disappears altogether at the compressed spot. Above, and below, it retains its natural thickness, though, probably, only for a certain time. This form of atrophy evidently affects the whole nerve equally, the nerve-tubes being at first diminished and compressed, and in the end absorbed.

It can be understood that inflammatory products in the neurilemma of a nerve may press upon the tubes and interfere with their nutrition, and so lead to their becoming atrophied: thus, while a nerve is actually increasing in size, its essential element may be diminishing.

3. A nerve may be atrophied in consequence of the wasting and loss of the peripheral or central organs to which it belongs, in fact, in consequence of a permanent solution of its continuity. Frequent examples of such atrophy may be adduced. On the one hand, the optic nerve shrinks because the globe of the eye is wasted, and the nerves diminish in size after the removal of a peripheral part by amputation; while, on the other hand, they are found to lessen in volume when their central organs have wasted; and, again, they are in an atrophied condition in hemicephalus, hydrocephalus, &c., at least within the skull. In these instances it is unquestionably the nerve-tubes which are affected; and all of them, or only a few are affected, according to the amount of wasting, or loss of substance at the periphery or the centre, which is the occasion of the atrophy. Under certain circumstances, nerves, which are extremely atrophied, acquire a translucid grayish-red appearance,

especially within the skull. The coloring is produced by the presence of a blastema, filled with numerous nuclei, which, at first gelatinous, and afterwards tough and elastic, takes the place of the nerve-tubes as they disappear, and becomes more distinctly visible as the original neurilemma of the affected nerve diminishes. The vessels of a nerve in this condition are often palpably dilated.

4. We not unfrequently observe a withered state of the ganglia, a shrinking and leather-like toughness of them, while their color has either altogether disappeared, or is changed to a rusty brownish, yeast-yellow, fawn, or slate gray. It appears frequently to be a primary affection; but in many cases it is a secondary consequence of previous disease. The chief example of it is the wasting of the abdominal ganglia, which follows typhus, and forms one of the few causes which can be found by the knife of the anatomist, for the sickly state succeeding typhus (Typhus-siechthum).

§ 4. *Solutions of Continuity.*—Nerves, just like other parts, are liable to incised and punctured wounds, to contusion, stretching, laceration, &c. A clean cut affords the best prognosis; especially when the ends of the nerve are secured against contact with the atmosphere, and pressure from excessive inflammatory tumefaction of other soft parts which may have been wounded at the same time; and when it is not irritated by foreign bodies, inflammatory products, &c. The process by which such wounds are healed and the nerve restored, have been studied both in man, and, by experiments, on animals. (J. Müller, and Sticker, Steinrück, Nasse, Günther, and Schön.)

After a nerve has been divided, its extremities retract a little, and some of the medulla is pressed out by the contractile neurilemmatous sheath. Inflammatory injection and reddening then ensue, with some swelling, which fills out particularly the looser cellular sheath of the fasciculi. Hence the fasciculi which compose each nervous cord are forced asunder at the divided extremity; and when exudation takes place, their separation is still more marked. The exudation is poured as well into the tissue of the neurilemmatous sheaths of the nerve, as into the space between its two ends; and coagulating there, it forms a gelatinous, yellowish-red mass, which reunites the divided portions, and mixes with the exudation furnished by the adjoining tissues which have been wounded at the same time. In this exudation nuclei and cells then form, and the regeneration of the nerve takes place. After some time, fibrils are perceived in the exudation, which advance towards each other, from the nerve-tubes of the two extremities; they are most distinct near the end of the nerve, and are least developed in the middle of the exudation-callus. After a still longer time, complete nerve-tubes present themselves in the callus, which appear on the whole smaller than the original tubes (Nasse); but some years later, old and new tubes can be no longer distinguished from one another. A different portion of the exudation serves for the regeneration of the neurilemma; whilst yet another portion remains behind, an unorganized (gestaltloses) blastema, and produces a nodular swelling of the new-formed piece. This nodule is generally largest at the upper extremity of the nerve, and does not disappear for a very long period. An adhesion forms between it and the cicatrix of the neighboring tissue; and this, too, may be equally permanent, or it may be loosened by absorption.

From the complete restoration of the function of the nerve, at least in all its material relations, it may be inferred that, most remarkably, in most, if not all, instances the proper primary filaments reunite. Sometimes this is not the case, as may be seen by anomalies in their direction; filaments alike in kind are united, but not those which properly belong to each other. Dissimilar nerves and filaments, such as motor and sensitive, seem never to unite with one another, at least their doing so is very questionable. (Bidder.)

Of course, the difficulties in the way of this regenerative process are enhanced by any loss of the substance of the nerve.

If no regeneration takes place, the primary filaments waste, especially those in its distal portion, and the whole nerve gradually acquires a grayish-red or gray aspect. In the other extremity these changes are confined to a certain tract, and to particular primary filaments.

The ends of the nerves divided in amputations swell out, and for the most part unite together in one common hard knot, with which the cicatrix of the neighboring soft parts also coalesces. If there be not soft parts enough to preserve the knot from pressure, or if the soft parts pull upon it, symptoms not unfrequently arise which may be so severe as to require that the nerves should be divided again.

Even the peripheral nervous bodies (ganglion-cells) appear to be capable of regeneration. (Valentin's Physiology.)

§ 5. *Diseases of the Texture of Nerves.*

1. *Congestion—apoplexy.*—It may be that nerves are not unfrequently congested; but there is scarcely ever opportunity in the dead subject to see any such condition of either their trunks or branches, except what has occurred since death.

More important instances of hyperæmia are found in the sympathetic system; its central ganglia becomes congested in the course of general acute processes of low type (dyscrasisch), which become localized in those structures to which the ganglionic nerves are distributed, especially in the mucous membrane and follicular apparatus of the intestines. The congestions of the ganglia just mentioned in the early stages of ileo-typhus, in the course of cholera and cholera-typhus, are of this class.

These congestions sometimes lead to apoplexy. In that case (during the same processes), the ganglia are found ecchymosed, or dotted with little round spots or streaks of extravasated blood, which are of about the size of a millet-seed.

2. *Inflammation.*—Inflammation may be observed in nerves as a consequence of injury, especially after they have been cut through: and in the trunks of larger nerves, such as the sciatic, rheumatic inflammation is met with in the dead subject. Inflammation may also pass over to nerves from adjoining tissues.

Its general characters are those of inflammation of fibrous tissue, inasmuch as its seat is the neurilemmatous sheath. When it is very intense, the characters of the red softening of nervous tissue are added. Its course is sometimes acute, sometimes chronic.

The marks of *acute* inflammation are as follow:

a. Injection, which presents a linear arrangement; and redness, which proceeds partly from the injection, and partly from small extravasations by which the neurilemmatous tissue is streaked and dotted.

b. Looseness, succulence, and *swelling* of the nervous cord, which result from the infiltration of serum into the tissue of the neurilemma, and into the sheaths between the primitive filaments. The fasciculi diverge manifestly from one another, and the nerve appears unravelled; and when the actual exudation is effused, this character is still more marked. The nerve has lost its smooth white glistening appearance; and its neurilemma is opaque, and appears rough and wrinkled.

c. Exudation. This is generally a grayish- or yellowish-red, gelatinous product, which sooner or later becomes firm. It is effused into the sheaths and tissue of the neurilemma, as well as between the primitive filaments themselves.

The intensity of these appearances varies widely. The redness may be a slight rosy tint arising from injection, or a deep saturated red color; it may be composed of some injected varicose branches, or of confluent ecchymoses. The swelling also may be of any degree, according to the quantity of the exudation. And further, the process may be merely confined to the outer layer of neurilemma, or it may extend more or less deeply to the sheaths of the several fasciculi.

d. The cellular tissue around the nervous cord always shares in these changes; it becomes injected, reddened, and infiltrated with a serous or sero-fibrinous brine-like fluid. The inflammation may even extend to the sheaths of neighboring muscles, to the fascia, subcutaneous cellular tissue, and general integuments.

This degree of inflammation may terminate quickly or slowly by *resolution;* or in *induration* of the nerve and a permanent abrogation of its function in whole or in part. The products of the process remain behind in the form either of a hardened blastema or of filamentous tissue; the nerve continues thickened, and more or less misshapen, and forms a grayish cord, which is sometimes marked with black pigment, and crossed by varicose vessels; the fasciculi coalesce, and the nerve itself adheres to the parts around. The nerve-filaments diminish in size and finally disappear, partly because of the pressure to which they are subjected by the exudation in which they are enveloped, especially as after a time it begins to contract; and partly because their nutrition is interrupted; for the vessels are obliterated by the inflammatory process, or are usurped by the exudation.

e. By higher degrees of inflammation and by a rapid and copious (tumultuous) exudation, the primitive filaments of the nerve are destroyed. They are found colored, and in a state of red, or of grayish- or yellowish-red softening, whilethe neurilemmatous sheaths have become easily lacerable. Such a condition may sometimes be seen in cases of spontaneous inflammation, but it is more common when the inflammation has succeeded injury. The exudation is generally purulent.

f. When this is the case, that is, when the exudation is *purulent*, the nerve appears highly discolored, and is infiltrated with a purulent fluid mixed with blood; its neurilemma is as fragile as tinder, and is losing its vitality, while the mass of the nerve is changed into a yellowish-red, brownish-red, or chocolate-colored pulp. The cellular tissue around the nerve becomes infiltrated with yellow fibrinous exudation, and abscesses of various size are formed here and there in its course.

The next step in such an inflammation is an *ulcerative* destruction of the nerve. But should it stop short at this point, granulations appear, which become progressively changed into cicatrix tissue; just as is observed in the stump of a nerve after amputation. It must, however, be remarked that the nerves resist, for a long time, the suppurative and sanious destruction which may be going on around them.

Chronic inflammation is characterized by the varicose state of the vessels of the affected nerve, by products which become indurated, and gradually increase in quantity, and by a change of the nerve to a slate- or lead-gray color. Sometimes the products are not deposited uniformly throughout the nerve, and then nodular swellings are formed on it.

There is one appearance which I must here mention as an appendix to the foregoing descriptions; it is the following condition of the nerves in cases of traumatic tetanus: and, however insignificant and unsatisfactory it may seem when brought into connection with the symptoms during life, yet it is the only real fact that has been made out in such cases after death. Froriep has ascertained that, besides the inflammation which is seen in the nerve at the spot which has been injured, a rosy reddening is produced at irregular intervals in its course by the injection of its neurilemma, but it is unaccompanied by any distinguishable products. The reddening is mostly confined to the surface of the nerve, though it sometimes dips a little way between the fasciculi. If a plantar nerve, for instance, has been injured, it is repeated three, four, five, or more times in the course of the tibial and sciatic nerves up to the sacral plexus; but neither where these nerves enter the medulla nor in the cord itself is any similar appearance to be found.

3. *Morbid growths in nerves.*

a. Cysts.—Even in the trunks of very large nerves such formations are extremely rare. It is at once clear from the conditions under which the cyst is developed that it performs the office of a bursa.

b. Fibroid tissue.—This structure is met with in nerves chiefly as a product of inflammation. But there are also certain tumors which are known generally by the name of neuroma, and which appear to me to rank with fibroid structures. When they exist in great number, as they sometimes do, pervading many, or most, or even all of the nerves, they constitute that which Serres has most erroneously denominated a change of the nerves to ganglionic structure.

Neuroma is the most frequent adventitious growth, except cancer, which occurs in the peripheral nervous system; and cancer is very frequently only a secondary affection in which nervous tissue is involved in consequence of its contiguity to other diseased organs.

Neuroma forms round, or more usually elongated and oval, tumors, the long diameter of which is parallel to the nerve. They are of a tough elastic consistence, and of a grayish or pale yellowish-red color, and are invested with a distinct fibrous sheath. In size they vary from a scarcely discernible enlargement of the nerve, to the bulk of a walnut or even of a hen's egg, and in number, from one until they are almost countless. In this last case neuroma constitutes a very remarkable general disease, as to the cause of which we are still in the dark. It has already been observed several times, and three times in the Vienna Hospital. It has been noticed in such cases, that the size of the tumor bears no direct proportion to the thickness of the nerve on which it is formed, for in fact those tumors which are situated on small branches are relatively much larger than those which occupy the thick trunk of a nerve.

The tumors lie between the fasciculi of the nerve and are interwoven with their neurilemmatous sheath. And it is a remarkable, and no less important general rule, because of the symptoms which may result from its presence, or which may be set up by operations performed on it, that neuroma is never deposited in the centre of a nerve, but at its side, so that only a small part of its fasciculi is displaced; the displaced fasciculi are spread abroad and stretched over the tumor, while the greater mass of the nerve remains on the other side uninjured, and with its fibres in connection with one another.

Neuroma occurs most frequently in the spinal nerves and in those cerebral nerves, motor as well as sensitive, which resemble the nerves of the cord: it may occur at any point in their course, even within the canal of the dura mater of the cord, and close to their junction with the medulla.

Although neuroma, when a solitary tumor, frequently occasions very considerable annoyance and pain, yet such is by no means invariably the case; and in those instances in which neuromatous growths were extensively diffused through the nervous system they had given rise to no symptoms whatever.

c. I have never met with any instance of anomalous bony substance, or so-called *ossification*, in nerves, although the callus remaining in them after inflammation, and neuromatous tumors, seem to furnish a suitable foundation for the production of it.

d. Nerves may be destroyed in the softening of *tubercle* either around or adjoining them, though the larger nerves resist tubercular and all other suppurative destruction for a long time. Nerves are never the seat of primary tubercle. With cancer, however, the case is different.

e. *Cancer* often occurs in the peripheral nervous system as a secondary disease, the nerve yielding to the aggression of a neighboring cancerous growth: and not unfrequently it presents itself as a primary disease. In respect to the former, the secondary affection,—when any organ is the seat of cancerous degeneration, the nerves included in it may be destroyed in the same manner as other tissues; or a cancerous mass, as it advances, may involve any nervous trunk that lies upon it.

Whether the nerve be primarily or secondarily diseased, the form of the cancer is generally the medullary; indeed, the white medullary cancer and melanosis appear to be the only forms with which nerve is primarily affected. Although a nerve when secondarily affected with any kind of cancer, is attacked, as a general rule, in the same manner as any other tissue, yet it is remarkable how little resistance it appears to offer to the inroad of medullary cancer in particular.

Primary cancer may be developed at the peripheral extremity of a nerve, or at any other spot in its course, up to its termination in the nervous centre. The former is by far the most frequent situation, and the best known example of it is medullary cancer of the retina.

Medullary cancer of nerve, as that of the retina shows, commonly reaches a very large size. Very often it is quite alone in the system, though not unfrequently it is combined with cancer in other organs. Thus cancer of the optic nerve within the skull, and of the brain, often coexists with cancer of the retina. The extirpation of *fungus bulbi*, or primary cancer of the retina, is, as a general rule, succeeded by a very rapid and extensive (tumultuous) development of cancer in several organs at once.

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ROKITANSKY'S

PATHOLOGICAL ANATOMY.

VOLUME IV.

A MANUAL

OF

PATHOLOGICAL ANATOMY.

BY

CARL ROKITANSKY, M.D.,
CURATOR OF THE IMPERIAL PATHOLOGICAL MUSEUM, AND PROFESSOR AT THE
UNIVERSITY OF VIENNA, ETC.

VOLUME IV.

THE
ORGANS OF RESPIRATION AND CIRCULATION.

TRANSLATED FROM THE GERMAN,

BY

GEORGE E. DAY, M.D., F.R.S.,
FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS;
PROFESSOR OF MEDICINE IN THE UNIVERSITY OF ST. ANDREWS, ETC.

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PART I.

DISEASES OF THE RESPIRATORY SYSTEM.

PART I.

ABNORMAL CONDITIONS OF THE RESPIRATORY ORGANS.

We shall consider the abnormal conditions of the respiratory organs under three heads:—

1st. As they occur in the air-passages; namely, the larynx, trachea, and bronchi.
2d. As they occur in the pleuræ; and—
3d. As they occur in the lungs.

The deviations from the normal state occurring in the thyroid and thymus glands will be noticed in a Supplement.

I.—ABNORMAL CONDITIONS OF THE AIR-PASSAGES.

§ 1. *Deficiency and Excess of Formation.*

An entire deficiency of the air-passages invariably occurs in cases in which the lungs are absent. A partial deficiency, as for instance of the trachea, may occur without the lungs being necessarily absent, the bronchi in such cases being given off directly from the larynx. Under this head we must notice imperfect development of the air-passages, dependent on the deficiency of certain parts entering into their structure, as of some of the laryngeal cartilages or tracheal rings; and their malformations arising from the arrest of development both in length and width.

An excess of formation is seen in the duplication occurring in double monsters, in which either the upper or the lower portion of the pulmonary apparatus is doubled; and here we must also place the occurrence of a supernumerary third bronchus, occasionally noticed on the right side in persons otherwise normally developed. Finally, we sometimes meet with supernumerary laryngeal cartilages and tracheal rings, thereby increasing the distance to the tracheal bifurcation.

§ 2. *Deviations in Size.*—In noticing the *calibre* of the air-passages, we shall omit the consideration of those cases in which the whole apparatus is either extremely developed, or where, on the other hand, it appears in an undeveloped condition with its walls in an attenuated state, and shall at once proceed to the subject of *acquired dilatations and contractions.* In noticing the *thickness of the walls* of the air-passages, we shall have to consider the *hypertrophy* and *atrophy* of the various structures entering into their composition.

a. Morbid Dilatations of the Air-passages.—These occur in variously recurring forms in the larynx, trachea, and bronchi, and sometimes occasion a dilatation of the *whole* apparatus; much more commonly, however, the dilatation affects only *single portions* of the respiratory organs, as, for instance, the bronchi, where, by the way, this morbid change is most frequently noticed.

1. *Dilatation of the Larynx and of the Trachea.*—A uniform dilatation of this canal is not unfrequently seen in *marasmus* or *senile atrophy*. Its existence in advanced age is interesting, since it always occurs with senile marasmus of the lungs (emphysema senile), and is more or less proportional to it. They are both dependent on the wasting of the tissues entering into the formation of the larynx and trachea.

There is another form of dilatation, which proceeds from *hypertrophy and relaxation of the posterior wall of the trachea, with or without saccular or hernial protrusion of the mucous membrane.* This form is extremely rare in the larynx, as, indeed, might have been naturally expected from the protected state of the interstices between its different cartilages; in fact, we scarcely ever see even a tendency towards it: while in the trachea it is of frequent occurrence, and is sometimes developed to an astonishing degree. Although bronchial dilatations have been well understood since the time of Laennec, little has been observed in reference to dilatation of the trachea, and especially in regard to this form.[1]

In the first place, there is a relaxation of the posterior wall of the trachea, giving rise to a great augmentation of surface, especially in the lateral directions. Moreover, its mucous membrane, transverse muscular fibres, and mucous glands increase in bulk, and the excretory ducts of these glands become dilated; while, on the other hand, the elastic, longitudinal fibres become attenuated and disappear. If protrusion of the mucous membrane should now occur, it gradually makes its way between the thickened transverse fibres in the form of a cleft or funnel, and finally of a transversely-placed, saccular expansion, usually deepest at the posterior part of the tracheal rings, where we find a distorted, cleft-like orifice of the excretory duct of a mucous gland. The larger this hernia or false diverticulum becomes, so much the more prominently do the muscular bands, which limit it, project on the inner surface of the trachea; and here, if the herniæ be numerous and close upon one another, the muscular fibres form a lattice-work, in which the cross-bars are usually single, but occasionally bifurcated at one of the extremities.

This condition is dependent on repeated and chronic catarrhs of the trachea, and forms *one* of a number of analogous cases occurring in other parts of the body. In saccular dilatation, the hypertrophied mucous glands on the posterior wall of the trachea, by the traction through the medium of their ducts, draw the tracheal mucous membrane between the bundles of transverse fibres. These dilatations sometimes extend along the whole trachea, and even into the bronchi.

These dilatations of the trachea closely correspond with the similar dilatations of the bronchi, proceeding from hypertrophy and paralysis.

2. *Dilatation of the Bronchi (Bronchestasis).*—There are forms of bronchial dilatation besides that which depends on the wasting of the tissues

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[1] See Oesterr. Jahrb. vol. xvi. p. 3.

in old age, or senile marasmus. In fact, this portion of the air-passages is remarkable for the frequency with which dilatations occur, and for the degree of development which they attain. They constitute one of the most important diseases of the air-passages.

There are *two forms* of bronchial dilatation which especially claim our attention:

a. In the *first* we find a bronchial tube *uniformly dilated* through a certain extent; that is to say, the dilatation has taken place uniformly at all points of its periphery, so that a tube, which in the normal state will admit only of a fine probe, will now admit of the passage of a crow or goose-quill, or even of a larger body. The dilatation is very striking and distinct, when we see a bronchial tube far exceeding in size the stem from which it is given off. It is seldom confined to a single tube, but, as a rule, affects a distinct portion of the bronchial tree; and its branches and twigs may either undergo an augmentation proportional to their relative natural sizes, or, as is more frequently the case, the dilatation becomes more considerable the deeper and further we proceed. In this it observes a law to which we shall frequently recur.

β. The *second* form is the *saccular dilatation.* Here we find a bronchial tube dilated into a fusiform or roundish sac; the dilatation in the latter case very frequently preponderating in such a direction, that the greater space of the bronchial sac lies altogether out of the axis of the tube entering or leaving it. These sacs, in rare cases, attain the size of a hen's egg, but most commonly they are of the size of a bean, hazel-nut, or walnut. We find, also, that either one or several bronchial tubes may undergo this saccular dilatation, while on both sides of the sac the normal calibre is retained, or the whole bronchial ramification may be affected. In the latter case, numerous similar sacs of various sizes are so arranged, that, collectively, they form a large ramifying sinuous cavity, whose individual excavations are bounded and separated from one another by ridge-like or valvular duplicatures, projecting inwards from the bronchial walls. Saccular dilatation of the bronchial extremities constitutes a special variety, which is frequently observed in the form of thin membranous vesicles, completely filled with air, and occurring, either singly or in groups, in the vicinity of cicatrizing tubercle in the apices of the upper lobes. One or more bronchial tubes, in taking their course through the impermeable substance of the apices of the upper lobes, crowded with obsolete and cretified tubercles, and, as it were, saturated with pigment, become compressed by the shrivelled parenchyma, and are finally obliterated; their extremities then expand into the above-mentioned vesicles; and, according to the state of the bronchial tubes—whether they are merely compressed or actually obliterated—the sacs may be emptied by gradual pressure, or will resist all attempts to expel the air.

Of these two forms of bronchial dilatation, the *second* appears to be the more common, and in young persons is undoubtedly the more frequent. The *degree* of dilatation may be determined by the proportion which it bears to the size of the bronchial tube, and to the calibre of the parent stem, from which it is given off. From the observations we have already made, it is obvious that, while its *extent* may be very limited, it may on the other hand be so considerable, that all the bronchial tubes of one lobe, or even of a whole lung, may be thus affected.

Bronchial dilatation occurs, for the most part, in the smaller tubes, and, as a general rule, is most frequent and, at the same time, most extensive, in those of the third and fourth order. It is never found in the two primary bronchi, or, at all events, very rarely, and then in the same form as the tracheal dilatations already described.

The bronchi near the surface and borders of the lungs are most liable to this affection; and this fact may be regarded as one of the evidences (and there are others) of the affinity between this affection and true vesicular emphysema of the lungs. The upper lobes are the most common seat of bronchial dilatations.

The walls of the dilated bronchi are found in various conditions. Sometimes we observe the mucous membrane and the fibrous sheath *hypertrophied* and *thickened*. The former appears in a state of chronic catarrh, being tumid, of a more or less dark-red tint, of a loose spongy appearance, and permitting of being easily torn. The bronchi are rigid; on making a section of the lung, they appear like wide, gaping tubes from which a thick, yellow, purulent mucus is seen to flow, and their white, thick fibrous sheaths strongly contrast with the inner layer of tumid and reddened mucous membrane. Such is the usual character of the first form of bronchial dilatation.

In the saccular form of dilatation, the walls are *relaxed* and *attenuated*. The mucous membrane of the bronchial sacs is only slightly, or not at all, reddened; it is more commonly pale; the firmness of its tissue is very little, or not at all, modified; and it generally presents a smooth and polished appearance, similar to that of a serous membrane. The sacs contain a thin, pale-yellow, puriform fluid, or an almost colorless vitreous mucus.

From the very striking and almost constant differences presented by the bronchial walls in these two forms of dilatation, we are led to infer that there are corresponding differences in their nature and their causes; and we shall presently have an opportunity of pointing out in what these differences actually consist.

The pulmonary tissue surrounding a bronchial dilatation is generally of increased density, and finally becomes obliterated. We shall subsequently enter fully into the consideration of this change, and the conditions giving rise to it; and shall point out its importance in the establishment of a theory relating to the production of bronchial dilatation.

Laennec, and almost all subsequent pathologists, believe that bronchial dilatation is always a mechanical consequence of catarrh; that it is dependent on the accumulation of the bronchial secretion at certain points, and the powerful inspirations made during paroxysms of coughing, whereby the walls, from some unknown causes, become at one time thicker, and at another thinner, than natural. The condensation of the surrounding parenchyma is, according to their views, simply dependent on the compression exercised by the dilated bronchus, which thus becomes the primary cause of the final obliteration of the pulmonary tissue. Hence, according to Laennec, the bronchial dilatation is the primary anomaly, and the condensation of the pulmonary substance is merely a secondary change dependent on its compression by the tube.

Corrigan has recently published a theory to account for bronchial dilatation, which is directly the opposite to that of Laennec; and believing the disease to be allied, in its anatomical elements, to cirrhosis of the liver, he has given it the name of *cirrhosis of the lungs.* He regards the atrophy and obliteration of the parenchyma of the lung as the *primary* phenomenon in the development of the disease, and as occurring spontaneously, while the dilatation of the bronchi is *consecutive*, and is not only dependent on the tendency to fill the space thus rendered vacant, and on the expansion occurring in the act of inspiration, but also on the traction exerted on the opposite walls of the bronchial tubes, by the shrinking of the surrounding tissue.

To speak more precisely, the change that the parenchyma of the lung surrounding bronchial dilatations undergoes, consists, in extreme cases, of obsolescence and destruction of the cellular spaces, and of the contraction of the cells so as to form a cellulo-fibrous or even a callous and fibro-cartilaginous tissue, which may be either white or streaked with blackish-gray pigment, dotted or of a uniform color, and which is so intermixed with the fibrous sheath of the bronchus that the two form one continuous whole.

When we take into consideration the vast extent to which the parenchyma of the lung around a dilated bronchus is obliterated; when we reflect on the nature and the degree of this metamorphosis, which very rarely follows prolonged external pressure on a lung; and, finally, when we notice the circumstance that this metamorphosis does not always develope itself uniformly around the dilated tube, nor is most marked the nearer we approach it; we cannot help doubting if all this can be produced and explained by the mere pressure caused by the dilatation of a thin membranous bronchial tube.

In point of fact, on instituting a closer examination, we arrive at conditions of another kind, which are in themselves sufficient to explain this metamorphosis, and are of the greatest importance in reference to the genesis of bronchial dilatation.

Whichever be the form under which bronchial dilatation appears, bronchitis must be regarded as the most frequent primary cause. It acts in different ways, but not mechanically, from accumulation of mucus, according to the theory of Laennec.

In the *first* form of bronchial dilatation, as we have described it in a preceding page, atony and paralysis of the contractile and irritable elements of the tubes are present, dependent on chronic inflammation and blennorrhœa. The facility with which the walls undergo dilatation through the influence of the inspirations and the concussion induced by the paroxysms of cough, is proportioned to the amount of exertion rerequired to throw off the secretion accumulated in the bronchial tubes. Moreover the circumstance that many of the smaller tubes are completely obstructed by the blennorrhœal secretion, favors the above condition. This form of bronchial dilatation affects that portion of the bronchial system in which blennorrhœa occurs.

The *second* or saccular form of bronchial dilatation is not developed in that portion of the bronchi which is the seat of catarrh, but beyond it; it is the consequence of bronchitis in the final ramifications of the bronchi, and depends on their obstruction by the accumulation of secretion, on the tumid state of their mucous membrane, and finally on their actual obliteration. It is produced by the hinderance that is presented to the free ingress of the inspired air, and is proportional to the difficulty of breathing (Reynaud), and the prolonged length of each individual inspiration; and it is especially developed in and about the perfectly impermeable bronchial tubes. The parenchyma surrounding this portion of the bronchial system collapses, and thus produces a space which becomes filled by the dilating bronchus. The dilatation thus lies entirely, or for the most part, in a collapsed and apparently compressed portion of parenchyma; hence, the latter appears to be the *primary* anomaly, and the bronchial dilatation merely a *resulting* and consecutive morbid change. This circumstance, together with the fact that the collapsed parenchyma passes into the above-described state of complete obliteration, and thus as it were, contracts upon itself and causes additional space in the lung, closely approximates this theory to that of Corrigan.

According to him the primary affection is not bronchitis, but is a disease of the parenchyma, not so much presenting the characters of inflammation of the interstitial areolar tissue, as of a pneumonic process (of which we shall subsequently treat) insidiously extending itself from one lobule to another, and depositing a product which becomes indurated, and fused or blended, as it were, with the original tissue. The air-cells become thus obliterated and destroyed, and undergo the same change. The resulting cellulo-fibrous tissue may here, as in the first case, draw asunder the bronchial walls by the tension induced by its further contraction, and may thus contribute to the further dilatation of the bronchial sac and to the increased attenuation of its walls.

However this may be, a smaller or larger proportion of the lungs always becomes obsolete and shrivelled in proportion to the extent of the bronchial affection; indeed, when the bronchial tubes of a whole lung are thus affected, we find that all of its parenchyma is more or less obliterated, contracted to a small part of its normal volume, as if in consequence of external pressure from exudation, and drawn up in the mediastinum towards the bronchus; moreover, the cavity of the thorax is diminished, in consequence of the sinking of its walls over the cellulo-fibrous tissue surrrounding the dilated bronchial tubes.

In some rare cases a bronchial sac is entirely separated by obliteration, not only from its own branches, but also from the tube on which it was situated. It then exhibits the appearance of a perfectly closed cavity, which, in consequence of the persistent secreting activity of its lining membrane, is probably further enlarged by the accumulation of mucus. Subsequently, however, if this secreting action be suspended, the accumulated matter becomes inspissated and diminished in volume, till ultimately there is nothing left but a fibrous capsule, enclosing either a soft, fatty, calcareous mass, or a solid concretion of bone-earth.

Isolated saccular dilatations containing puriform matter may be mistaken for *tuberculous cavities*, especially when they are associated with pulmonary tubercles and are situated in the upper third of the superior lobes of lung,—the ordinary focus of pulmonary tuberculosis. On a closer examination we may recognize a saccular bronchial dilatation by the roundish form of the cavern and its pouches, by the smooth, uninjured, investing mucous membrane, by the absence of all signs of ulceration in the bronchial tubes entering it, by the striking difference between its contents and tuberculous pus, by the circumstance that the surrounding and contiguous obsolete parenchyma contains no tuberculous granulations, or only such as are obsolete, and by the simultaneous occurrence of similar cavities in parts of the lung, in which tuberculous excavations are not usually found. In other cases our diagnosis must be founded on general principles, as for instance, on the fact that bronchial dilatations are ordinarily found in the superficial parenchyma of the lungs towards their borders, and very rarely occur in the summit; and further, that, when the bronchial disease is very extensive, tuberculosis is incompatible with it.

In consequence of the obliteration of a large extent of lung produced by extensive bronchial dilatation, we find that this affection gives rise to a development of the right side of the heart in the form of active dilatation, stasis and dilatation of the whole venous system, cyanosis, and vicarious development of the permeable portions of the lungs, which not unfrequently leads to bronchial and pulmonary hemorrhage (hæmoptoic infarctus). If the bronchial dilatation be very highly developed, it induces collapse, emaciation, a cachectic appearance, dropsy, and finally total exhaustion.

In consequence of the venosity and cyanosis to which it gives rise, it affords a very striking immunity, not only from pulmonary tubercles, but from tuberculosis in general. The fact that bronchial dilatation exerts an excluding influence on pulmonary tuberculosis has been known since the time of Laennec; and although the reasons for this influence are not understood, it has served, in recent times, as the basis of several plans for the cure of pulmonary consumption.

b. Contraction of the Air-passages.—This may occur in any part of the respiratory apparatus; but the nature and degree of the affection may be extremely various; in fact, in the latter point of view, the change may proceed to closure and perfect obliteration.

1. It may be dependent on *external pressure.* There may be contraction of the larynx and of the trachea from an enlarged thyroid gland; of the trachea and bronchi from enlarged lymphatic glands, aneurisms, large cancerous deposits in the neck and mediastinum, enlarged thymus glands, and effusions into the cavity of the chest; and of the left bronchus by a dilated left auricle (King). In this manner the air-tubes become forced in various directions from their normal position, and their calibre, as may be seen in contraction of the trachea, may be so encroached on as to represent a mere fissure, having a transverse, an antero-posterior, a straight, or a crescentic form.

2. Contraction may be the result of *disease of the mucous membrane, or of the subjacent mucous tissue* of the air-passages, as of hypertrophy, inflammatory swelling, or œdema of the mucous membrane or of the submucous areolar tissue, of various excrescences, cancerous deposits, or cicatrices after loss of substance; the most frequent cause, however, is bronchitis, especially when it has given rise to obliteration of the finer bronchial tubes.

its diseases claim the most particular attention. They frequently extend to the subjacent tissues, and, for the most part, lead to their destruction.

A. *Diseases of the Mucous Membrane and of the Subjacent Areolar Tissue.*

a. Hyperæmia and Anæmia.—*Hyperæmia* of the air-passages is of comparatively common occurrence. When seated in the finer bronchial ramifications, it is combined with hyperæmia of the parenchyma of the lung; but in the larger bronchial tubes, and in the trachea and larynx, it usually exists alone, and independently of that affection. Hyperæmia varies extremely in importance, according as it is active, or simply mechanical and dependent on an obstructed circulation, or passive, which is a more rare affection; constituting, in any case, an independent disease, which finally gives rise to *hemorrhage*, or, under other conditions to *stasis*, and thus to acute or chronic *inflammation*. To the first belong the hemorrhages from the mucous membrane of the bronchi, trachea, larynx, and epiglottis. On examining the dead body, we find the air-passages to a certain degree filled with coagulated or fluid blood, and patches of the mucous membrane swollen, of a dark-red color, bleeding when pressed, and apparently loosened in texture; we find no other source of hemorrhage, no pulmonary apoplexy, nor mechanical or ulcerous separation of continuity. The lungs, as we find in other hemorrhages from the air-passages, present a dark or light-red speckled appearance from the deposition of blood in the terminations of the bronchial tubes and in the air-cells; but at the same time are (elsewhere) emphysematous, swollen, and pale, in consequence of the obstructed condition of the bronchial tubes, and the impediment thus presented to free expiration.

These hemorrhages occur in the active form during the period of evolution, and when there is general plethora, as vicarious to menstrual and hemorrhoidal fluxes. They arise from and accompany the congestions which so frequently precede the development of tubercles in the lungs, and may be produced by any strong exertion, but especially by the overtaxing of the respiratory organs. They may arise from any sudden shock to the lungs, from the sudden rarefaction of the atmosphere, and they are very frequently dependent on mechanical hyperæmia, resulting from hypertrophy and dilatation of the heart.

Anæmia of the mucous membrane of the air-passages is more or less developed in senile and other varieties of atrophy.

b. Inflammations of the Mucous Membrane.

1. *Catarrhal Inflammation.*—This is one of the most common diseases of the air-passages. It may be either *acute* or *chronic*, and in one or other of these forms often attacks only single portions of the whole bronchial apparatus. In the acute form, however, it not unfrequently extends over the whole bronchial tract, and in the chronic form also is sometimes as widely diffused, but in this case the inflammatory action is more intense at some points than at others.

We distinguish, according to their positions, *catarrhs of the larynx, trachea, and bronchi;* or *laryngitis, tracheitis, and bronchitis catarrhosa.*

α. *Acute Catarrhal Inflammation.*—This presents the same anatomical appearances in whatever part of the bronchial system it occurs; there are various degrees of redness, relaxation, and swelling of the tissue, which, according to the intensity and stage of the affection, secretes a diminished or increased amount of muco-serous, frothy fluid (sputum crudum), or thick whitish or yellowish puriform mucus (sputum coctum), or, finally, of true purulent matter (the transition to superficial suppuration). The swelling of the mucous membrane and the submucous tissue, which assume the form of watery infiltration, from the areolar tissue being accumulated at individual spots, is important and worthy of great attention, on account of the facility with which it interferes with the calibre of the tubes. This swelling of the mucous membrane is most dangerous when it affects the epiglottis, the folds limiting the glottis, the covering of the vocal chords, and the portion lining the ventricles: the danger is less, but still very great, when it attacks the smaller bronchial tubes.

Acute bronchial catarrh, when widely diffused, is an important, and in children a perilous disease, not only in consequence of the contraction and perfect impermeability of the bronchial tubes caused by the swelling of the mucous membrane and the accumulation of its secretion, but from its occasionally, and especially in children, extending to the air-cells, forming catarrhal pneumonia.

β. *Chronic Catarrhal Inflammation.*—This is very frequent in certain portions of the air-tubes; it is often remarkable for its great intensity, and is of the highest importance from its sequelæ. These observations especially apply to chronic bronchial catarrh, but the affection is also common in the larynx and trachea, and sometimes extends over the whole course of the air-tubes; it is generally, however, then especially developed in some one particular part. It possesses the usual anatomical characters of chronic inflammation of mucous membranes; but as no acute catarrh is so liable to frequent relapses, and exhibits such a tendency to become habitual as that affecting the air-passages, so also the chronic form is here especially liable either to relapse into acute inflammation, with an augmentation of intensity, or, on the other hand, to degenerate into *blennorrhœa.* It gives rise to swelling of the mucous membrane, especially on those parts of the larynx which we have already described as abounding in glands, thus causing *glandular hypertrophy*, *mucous polypi*, and .*cauliflower epithelial growths;* similarly in the trachea, and more especially in the bronchi, it causes a *spongy thickening* of the mucous membrane; and these affections may lead to *hypertrophy and relaxation of the submucous muscular strata, of the fibrous portions of the vocal apparatus, and of the fibrous sheaths of the bronchi*, and sometimes to *ulcerous destruction*, especially of the larynx, in the form of *diffuse catarrhal suppuration*, or of *catarrhal follicular ulceration.*

Chronic catarrh may further give rise to *diminution* of the calibre of the air-passages, amounting even to their perfect closure; the previous loss of substance sometimes inducing adhesion and perfect *obliteration* of the bronchial tubes. At other times, associated with hypertrophy and paralysis of the tissues, it gives rise to bronchial *dilatation.*

The quantity of whitish, cream-like, or yellow purulent secretion that is thrown off by the bronchial mucous membrane in a state of blennorrhœa, is very remarkable, especially where dilatation is at the same time present. The cases of what is termed Phthisis pituitosa (asthma humidum, bronchial blennorrhœa), fall under this head; and on making an incision through the lungs, abundance of mucus is seen gushing out of the divided bronchi, and pouring over the cut surface.

Both the acute and the chronic form of pulmonary catarrh, may occur as isolated and substantive diseases; they are, however, frequently associated with catarrhs of other mucous membranes. The acute is frequently of an exanthematous nature, and seems especially connected with measles, small-pox, and typhus; while the chronic form is often of a gouty, scrofulous, or syphilitic nature, and is associated with the most different pseudo-plastic processes on the mucous membrane, and in the submucous tissues. It is the chronic form of bronchial catarrh which accompanies pulmonary tuberculosis, especially true tuberculous phthisis. Moreover, it very frequently arises from mechanical hyperæmia induced by cardiac diseases.

Gonorrhœal catarrh of the larynx requires especial notice, in consequence of its sequelæ. In the form of gonorrhœal metastasis, it attacks the mucous membrane of the epiglottis, and the lateral duplicatures of the glottis and of the superior vocal chords, converting the mucous membrane and subjacent areolar tissue into a fibro-lardaceous, white, resistant structure of considerable thickness, thus giving rise to contraction of the rima gottidis and the cavity of the larynx. This constitutes *gonorrhœal stenosis of the larynx.*

There are two distinct modes in which chronic bronchial catarrh acts injuriously on the parenchyma of the lung. It sometimes causes emphysema; at other times collapse and obliteration of the air-vessels, and consequently obliteration of the pulmonary tissue itself. Amongst its sequelæ we may mention livor, cyanosis, active dilatation of the right side of the heart, and hydrothorax; and the patient dies asphyxiated through some of these affections, or sinks from tabes under the form of Phthisis pituitosa.

2. *Exudative Processes* (*Croupous Inflammation*).—Under this head we must place processes allied to each other, since they originate in one general disease, but differ extremely in their local morbid centres. This difference exhibits itself anatomically in the physical qualities of the inflammatory products on the free surfaces of mucous membranes, and in the condition of the mucous membrane itself, and of the submucous areolar tissue. These processes, especially in true croup, are primary and independent; or in their collective forms they may be secondary affections—the evidence of a degenerated acute or chronic disease.

True *croup, the exudative process yielding a plastic, fibrinous product,* claims our first attention. We scarcely ever observe the primary, genuine croupous process to occur anywhere except on the mucous membrane of the air-passages, where it appears as *laryngeal, tracheal,* or *bronchial croup,* ordinarily known as *laryngitis, tracheitis, bronchitis polyposa seu membranacea.* It not unfrequently extends over the whole of the air-passages, from the epiglottis to the minute ramifications of the bronchial tubes; and often affects the throat and pharynx, and sometimes even the œsophagus. It either attacks extensive continuous tracts of mucous membrane, or confines its ravages to isolated patches, and hence the exudation or croup-membrane either presents the appearance of continuous, tubular, arborescent coagula, corresponding with the division of the trachea and the bronchial ramifications, or of irregular patches, as is most commonly observed on the larynx. In bronchial croup, the tubular exudations from the larger bronchi present a calibre inversely proportional to their thickness, and those thrown off from the finer ramifications occur as solid cylinders.

The exudations present great differences in thickness and consistence; the membrane sometimes resembling an investment of hoar-frost, or gauze, whilst at other times it will even exceed a line in thickness, while the consistence may vary from that of viscid cream to that of the most compact tough, leathery coagulated fibrin. But neither the density nor the consistence is generally uniform throughout; the exudation, as a general rule, becomes thinner and gradually softer towards its edges, more puriform or creamy, and the portion in contact with the mucous membrane is the softer and looser of the two.

In color they are yellowish-white, or gray, and not unfrequently have a greenish tint; they either adhere firmly to the mucous membrane, or hang loosely on it, the latter being the case when a viscid secretion occurs between the false membrane and the mucous surface. The surface next to the mucous membrane is frequently marked with red streaks and dots, consisting in part of blood adhering to the surface, and in part, as found on closer examination, of straight or tortuous vessels, or of small, roundish extravasations, from which currents of blood are seen to emerge in an arborescent and radiating form. The appearance of the subjacent mucous membrane is liable to considerable differences; its red color sometimes assumes a very dark, almost brown tint, but more frequently a bright erysipelatous hue; and again it occasionally, but very rarely, happens that all the signs of injection are absent; it presents an appearance of sores, as if it were excoriated, bleeds from numerous, minute, scattered spots, and presents various degrees of swelling. The swelling is, however, sometimes so very trifling as hardly to attract notice. The submucous areolar tissue is most commonly, if not always, the seat of serous infiltration.

Genuine croup of the air-passages is essentially a disease of childhood; it rarely, however, occurs before the end of the second year, and the parts it most commonly attacks are the larynx and trachea; in adults, bronchial croup is the most common variety, and during the age of puberty and early manhood, it is often associated with pneumonia. Croup of the final ramifications of the bronchi occurs simultaneously with pneumonia, and usually runs an acute course; sometimes, however, it assumes a chronic form, the process continuing with less intensity for a longer period, with occasional exacerbations, which give rise to the deposition of fresh products. In many persons it becomes habitual, and often, in the form of *bronchial croup*, seems to assume a certain degree of periodicity in its attacks. It is frequently combined with pneumonia, pleurisy, and pericarditis, and sometimes with meningitis, and acute and chronic hydrocephalus; and it occasionally extends to the stomach and degenerates into acute softening of that organ. It proves fatal from the contraction which it induces in the air-passages through exudation, and still more through the swelling of the mucous membrane over the subjacent areolar tissue, and from spasmodic closure of the glottis; moreover suffocation is frequently induced by *pulmonary œdema*, or the patient may occasionally sink from the exhaustion induced by very abundant exudation. We have no anatomical evidence that the pneumogastric nerve is seriously affected.

The other exudative processes yield a soft, purulent, and less plastic exudation, or a thin, sero-purulent, gelatinous, discolored ichor, which attenuates, and finally dissolves the mucous membrane. The submucous areolar tissue is infiltrated by a matter of a similar character, and its texture is rendered friable, lacerable, and fusible. These are, in most cases, secondary processes, depending on the localization of a degenerated general disease of an acute exanthematous nature,—as variola or scarlatina.

All the exudative processes on the mucous membrane of the air-passages are frequently combined with similar processes on other mucous or serous membranes; they may degenerate into gangrene and acute softening, and from the development of the spleen, lymphatic glands, and follicular apparatus of the intestinal mucous membrane in these cases, we conjecture that they originate in a disease of dyscrasia of the whole mass of the lymph and blood.

Here we must also notice aphthæ of the air-passages; they are for the most part confined to the larynx, trachea, and the great bronchial trunks, very seldom extending to the throat; they scarcely ever occurs as a primary affection, but are most commonly associated with tuberculous phthisis of the larynx and the lungs.

3. *Pustular Inflammation.*—The only form of pustular inflammation occurring in the air-passages is the *variolous*, which, however, is very perfectly developed. It is usually present whenever the variolous process exhibits considerable intensity, and when the skin is covered with an abundant eruption of the exanthema. It appears in the form of simple pustules on the mucous membrane of the epiglottis and adjacent soft palate, of the larynx, and the trachea, and not unfrequently in the bronchi and their primary branches. The pustules are soft, easily rubbed off, not unfrequently confluent, and when removed leave a superficial, concave, roundish spot, where the mucous membrane presents a dark-red or livid tint, and an appearance of excoriation. Between these spots it presents various degrees of redness and thickening, and is coated with a tough plastic mucus,—a croupous exudation; moreover it is much swollen, and together with the submucous areolar tissue, exhibits signs of serous infiltration. Very intense confluent pustulation may give rise to *variolous ulceration.*

4. *The Typhous Process on the Mucous Membrane of the Air-Passages.*—The typhous process occurring in the air-passages presents numerous peculiarities in reference to its connection with the general disease, with the morbid state of the mucous membrane of the small intestine, where amongst us it usually becomes localized as ileo-typhus, and in reference to its seat generally.

In all cases of typhus it invariably occurs as a *typhous bronchial catarrh*, with tough and gelatinous-looking mucous. The catarrh seems to be developed in proportion to the intensity of the general disease, and is most severe in those cases which are marked by the predominance of catarrho-pectoral symptoms. It may occur here as the *true, special typhous process*, either in its genuine or its degenerate form; in this former case it may be either *primary* or *secondary;* in the latter case it is *always secondary*. Its seat is sometimes on the bronchial and at other times on the laryngeal mucous membrane; on the former it frequently occurs *as primary broncho-typhus*, and is a very serious affection; on the latter, constituting *laryngo-typhus*, it is almost always, at at least amongst us, a secondary process.

A. *Genuine typhus on the bronchial mucous membrane* always appears as an intense, diffused congestion; the mucous membrane is of a dark, almost violet tint, is swollen and succulent, and yields a secretion of a gelatinous and sometimes dark, blood-streak mucus, occurring in large masses. The disease is most commonly developed in the bronchial ramifications of the lower lobes; it is always limited to the stage of typhous congestion, and never gives rise to any apparent production of a secondary formation on the tissue of this membrane, such as is produced in immense quantity in the intestinal follicles in cases of abdominal typhus.

In *primary broncho-typhus* the general disease originally localizes itself here, avoiding all other mucous membranes, even that of the intestine, for which the typhous process in general shows the most decided preference; the latter mucous membrane exhibits, however, in many cases a recognizable, although always subordinate and secondary development of the follicles, in which the adjacent mesenteric glands participate; and in such cases it is very often a difficult matter to distinguish the typhus in the above-named affection of the bronchial mucous membrane. The peculiar stasis of the spleen and of the great *cul de sac* of the stomach, the remarkable intumescence of the former, and the singular character of the blood, the typhous nature of the general disease, and especially the altered condition of the bronchial glands, invariably serve, together with other symptoms, to indicate the typhous nature of the bronchial affection. The alteration occurring in the bronchial glands is of the same character as that affecting the mesenteric glands in abdominal typhus; they become swollen to the size of a pigeon's or even a hen's egg, are of a dark, violet color, which afterwards becomes lighter, present a relaxed and friable appearance, and are infiltrated with medullary typhous matter. Like typhous mesenteric glands they may become the seat of tumultuous metamorphosis, and thus, either with or without perforation of the adjacent mediastinum, may give rise to pleurisy.

This form is often combined with pneumo-typhus and typhous pleurisy, and is beyond all doubt the basis of the spotted contagious typhus, and very probably, also, of the Irish and North American typhus, which, in the majority of cases, run their course without any intestinal affection. With us this affection is rare, and, in point of frequency, is not to be compared to abdominal typhus.

Genuine secondary bronchial typhus presents the same anatomical characters, in a less highly developed state, as the primary. In a *degenerate* form it is very rare, occurring, for the most part, as bronchial croup, or as diffuse gangrene of the bronchial mucous membrane.

B. *Laryngo-typhus* is with us an unusually common and extremely unfavorable symptom in many epidemics in typhus. It scarcely ever occurs as a primary independent affection, but is almost invariably secondary, and forms, as it were, the completion of intestinal typhus, on various anomalies of which it is generally based.

It is almost invariably situated on the laryngeal mucous membrane above the transverse muscle, and towards the posterior extremities of the ventricles (a situation which, as we shall presently see, appears favorable to all the pseudo-plastic processes); it may, however, occur on the mucous membrane of the epiglottis, especially towards its lateral borders; and sometimes it occurs simultaneously at both these spots.

It, no doubt, frequently occurs in the *genuine* form, but it is only rarely that we have the opportunity of observing the typhous infiltration in its stage of crudity or of metamorphosis; as we see it in the dead body, there is almost invariably a loss of tissue, or ulcers of the same kind as those of the intestine, but less deep-seated.

Laryngo-typhus occurs, however, far more frequently in a *degenerate* form, either as an *exudative process* (croup), or more commonly as *gangrene*. The latter, after its detachment, leaves an ulcer, which cannot be distinguished from the degenerate typhous ulcer, so that we are unable from these appearances to draw any certain inference regarding the original process.

These ulcers are of a roundish shape, varying from the size of a lentil to that of a pea; they are either discrete or confluent, two or three often forming a group. They are seated at the spots we have already mentioned, on the posterior wall of the larynx and on the lateral edges of the epiglottis, on both of which situations they occur as linear ulcers; when, as is sometimes the case, they present themselves on the inferior surface of the epiglottis, they present a roundish or lenticular form; they are lax, discolored, and are black at the edges from the deposition of pigment; they gradually eat their way into the transverse muscle, the arytenoid and cricoid cartilages, the vocal chords and epiglottis, in which they give rise to softening, necrosis, and exfoliation. On the posterior laryngeal wall abscesses are not unfrequently developed, in which the necrosed arytenoid cartilages lie bathed in a brownish ichor; these abscesses sometimes penetrate into the pharynx. The whole constitutes a *typhous laryngeal phthisis.*

Laryngo-typhus is very frequently combined with pneumonia, and with secondary broncho- and pharyngo-typhus.

c. *Inflammation of the Submucous Areolar Tissue.*—In addition to the part that the submucous areolar tissue takes in inflammation of the mucous membrane of the air-passages, it is also subject to inflammations, occurring as primary affections. These inflammations are, however, rare, and for the most part of a metastatic character; and hence they have a special tendency to run into suppuration and necrosis of the areolar tissue and mucous membrane. In reference to their position and diffusion over the air-passages, they are usually limited to the submucous areolar tissue of the *larynx;* they may, however, extend to the corresponding tissue in the throat and pharynx, and even into the intermuscular areolar tissue of the neck. As results of *chronic* inflammation, we often in the larynx, meet with *hypertrophy*, *thickening*, and *callous induration of* this tissue, and a consequent *narrowing of the cavity of the larynx.*

d. Ulcerous Processes.—In the course of the preceding observations we have already noticed some of these processes; others still require to be described. Their position is, with very few exceptions, in the larynx and trachea, and as they are generally the result of a process originally proceeding from the mucous membrane, the direction of their destroying course is inwards into the tissues, and, as a rule, they make their way from within outwards.

Those we have already described are *catarrhal suppuration*, the *sloughing ulcer*, which, with the *aphthous ulcer*, must be regarded as degenerate exudative processes, the *variolous ulcer*, and the *typhous ulcer;* also the *suppuration and necrosis of the mucous membrane, proceeding from the submucous areolar tissue.* We have yet to consider *suppuration of the perichondrium with necrosis of the laryngeal cartilages*, and *tuberculous* and *cancerous ulceration.*

We must here notice syphilitic ulceration of the air-passages. Ulcers of this nature are, for the most part, situated on the epiglottis, having extended there from the soft palate and the root of the tongue. They usually present the characters of secondary chancres, and not unfrequently give rise to entire destruction of the epiglottis and of the mucous membrane around the glottis. The syphilitic destruction usually confines itself to these parts, leaving, after cure, a loss of part of the epiglottis, and thick, hard, white, tendinous, and cord-like cicatrices, crossing one another and giving rise to contraction. In rare cases, however, they extend to the larynx and trachea, and destroy the mucous membrane by causing sloughing and aphthæ, giving rise to contraction of the submucous tissue, and to friability and brittleness of the cartilages.

The walls of the air-passages are also liable to ulceration from without inwards. This occurs by far the most frequently in the bronchi, in consequence of their frequent proximity to softened tubercle and to tuberculous abscesses.

e. Œdema of the Mucous Membrane of the Air-Passages.—This affection has especially attracted the attention of pathologists when it has been situated in the larynx, where it has received the name of *œdema glottidis.* It is in this position that its attacks are most intense, and that its consequences are the most dangerous. In some few cases it extends to the mucous membrane of the posterior walls of the trachea and pharynx.

In the cases strictly falling under this head, it occurs as an infiltration of the submucous areolar tissue and of the mucous membrane itself, with a colorless or pale-yellow serum. When it occurs as œdema glottidis it is situated in the mucous membrane of the epiglottis, the duplicaturæ aryepiglotticæ, and the mucous membrane of the vocal chords and ventricles; and it constitutes a transparent pale-yellow, fluctuating tumor, which, in proportion to its size and extent, diminishes the aperture of the glottis, and may even entirely close it.

Œdema of the glottis, either in an *acute* or *chronic* form, may accompany not only all the inflammatory processes of the laryngeal mucous membrane of which we have spoken, but many other morbid conditions of the larynx and adjacent parts connected either essentially or incidentally with an irritation of the aforesaid mucous membrane. It accompanies catarrhal inflammations, especially those of an exudative nature, exanthematous processes, typhous and all ulcerous processes on the laryngeal mucous membrane, inflammations of the submucous tissue, tuberculous or cancerous affections of the larynx, &c.

These cases are of the highest importance, for the affection may become *rapidly* developed, and may cause death by asphyxia, in any of the above-named affections of the laryngeal or adjacent mucous membrane, as that of the velum palati or tonsils, even when the primary disease seems trifling; and it unfortunately happens that we are entirely ignorant of the peculiar conditions under which it is produced in these cases. Sero-purulent infiltration of the submucous areolar tissue may be confounded with true œdema; the former is, however, invariably the result of an intense inflammatory process.

f. Gangrene of the Air-Passages.—This affection occurs both here and in the parenchyma of the lungs in two distinct forms, either *as a circumscribed eschar* on the mucous membrane, eating its way into the submucous tissue, in which it may also occur primarily, or as a *diffuse gangrenous colliquescence of the bronchial mucous membrane.* The conditions under which it is developed are similar to those of gangrene of the lung, with which it is sometimes combined. It generally, however, occurs in tissues in some way previously diseased, but appears rather as an accidental termination than as a necessary consequence of any peculiar local morbid process. We have seen it take its origin from inflammation of the perichondrium of the laryngeal cartilages, from tuberculous laryngeal phthisis, from typhous ulcers, and laryngeal croup, and give rise to *circumscribed gangrenous destruction*, or much more frequently to *diffuse gangrene of the bronchial mucous membrane.* In the latter case, we find a certain extent of the mucous membrane either uniformly or at certain spots, of a dirty brownish-green color, and broken up into a soft, villous, moist, friable tissue, evolving the peculiar odor of sphacelus. The tubes are filled with a corresponding, discolored, frothy, stinking, sero-ichorous fluid. It is most commonly associated with pulmonary gangrene.

g. Adventitious Products.—See the remarks at *d*, page 35.

B. *Diseases of the Cartilaginous Skeleton of the Air-Passages.*

a. Inflammation of the Perichondrium of the Laryngeal Cartilages. (Perichondritis laryngea.)—In the examination of the dead body we have occasional opportunities of noticing a peculiar form of suppuration in the larynx, undoubtedly resulting from inflammation that had commenced in the perichondrium, which appears to be detached either at circumscribed spots, or more commonly over both surfaces of a whole cartilage, and under this a quantity of pus is found collected in a membranous sac. The cartilages are more or less denuded, rough, villous, necrosed, and perforated, or they lie entirely free, discolored, attenuated, softened, and more or less disintegrated in a large collection of pus. This abscess may make its way into the larynx, trachea, or pharynx, or may even open and discharge its contents externally.

This disease seems most frequently to attack the cricoid cartilage; it is commonly supposed to be of a rheumatic origin, and has been termed *rheumatic laryngeal phthisis;* it may, however, also occur as a consequence of the acute exanthemata (at least of variola) and of the mercurial disease.

b. Inflammation and Softening of the Epiglottis.—The epiglottis is sometimes the seat of a chronic inflammatory process, which finally leads to its conversion into a dense, rigid, fibro-cartilaginous tissue,—a change attended by shrivelling and deformity.

As a contrast to this rigidity, we may have softening of the epiglottis. This is probably also a result of inflammation, and is similar to the softening that occurs in the yellow coat of the arteries. The epiglottis loses its elasticity, becomes soft and friable, assumes a dirty yellow tint, and at length begins to waste away.

c. Ossification.—In the more advanced period of adult life, the cartilages of the larynx in the male are always more or less ossified; hence we need only notice those cases in which this change commences in early life at a more than ordinarily rapid course, or in which it spreads over an unusually great extent. The following is the order, in regard to frequency, of the parts thus morbidly affected; the thyroid cartilage, the cricoid cartilage, the tracheal rings, and the bronchial cartilages; it is extremely seldom that the arytenoid cartilages are affected. The ossification is here a true conversion of the cartilage into actual bone. The change may either occur spontaneously, or result from an inflammatory vascular activity in the perichondrium and cartilage, as is evidenced by its frequent occurrence at and below the seats of ulcers, especially those of a tuberculous character. Moreover, fractures and injuries of the cartilage give rise to this affection by inducing a deposition of ossifying callus. The newly-formed bone, in cases of laryngeal phthisis, may become the seat of caries and necrosis. Fragments of bone may then be expectorated, which, by the peculiar characters of their tissue, may be distinguished from various other earthy concretions that are sometimes ejected by coughing.

In certain rare cases we find the ossification extending not only to the most minute bronchial cartilages, but even to the finest of the membranous bronchial twigs. A system of rigid arborescent tubes then pervades the lungs, and hinders their collapse if a section be made through them; on passing the finger over the cut surface, a sensation is perceived similar to that produced by projecting, angular grains of sand. This change only occurs in very aged persons.

The *epiglottis* is never, strictly speaking, ossified, but we occasionally find that, when its texture has been modified by inflammation and has assumed a fibroid character, its form becomes variously modified, and it contains earthy deposits.

d. Adventitious Products.—Adventitious formations occurring in the air-passages are of the highest importance when they project into the interior of the air-passages in the form of broad or pedicled vegetations, and thus give rise to more or less contraction of their calibre. They occur almost exclusively in the larynx, and they are classified and treated of, according to their external characters, under the general head of laryngeal tumors. When considered in reference to their internal structure, they may be reduced to the following forms, which may be developed in and under all mucous membranes.

1. *Epithelial Formations.*—These occur in the form of roundish cauliflower or wart-like growths, varying from the size of a hempseed to that of a hazel-nut, and are occasionally even larger; they are attached by a short pedicle to the mucous membrane, present a somewhat lobular and laminated structure, and consist of exuberant epithelial cells and very delicate vessels, prolonged, as it were, from the mucous membrane. They are especially liable to occur on the vocal chords and arytenoid cartilages, but they are sometimes found on the under surface of the epiglottis, and on the cricoid cartilage. As they frequently spring from a cancerous basis, they are often of a malignant nature, but they have also been observed in a non-malignant form in persons of various ages after catarrhs and repeated attacks of croup. They are the most common of all laryngeal tumors.

2. *Cellular or Mucous Polypi and Condylomatous Excrescences.*—These occur upon or in immediate proximity with an ulcerated basis, or when there are no existing ulcerations, they form hard or spongy purple vegetations varying from the size of a pin's head to that of a hempseed or pea, lying sometimes in great numbers closely beside one another, and occupying large portions of the laryngeal mucous membrane. Their favorite seat is the mucous membrane of the vocal chords. They are most probably of a syphilitic nature, and when, as is sometimes the case, they are associated with tuberculous laryngeal phthisis, and constitute the tuberculous ulcer, we are led to suspect that they originate in a combination of the tuberculous with the syphilitic dyscrasia.

3. *Erectile Tissue* occurs in the form of broad-based, soft vegetations, capable of being rendered turgid, or as the development of the free extremities of mucous polypi. The former variety not unfrequently springs from a cancerous basis.

4. *Fibrous Tumors.*—These are extremely rare in the submucous areolar tissue of the mucous membrane lining the cavity of the larynx, but are more common beneath the pharyngeal mucous membrane investing the posterior wall of the larynx. They are here often found not only of their ordinary inconsiderable size, but sometimes of a very large volume, and by their adhesion to the pericardium, remind us of the large pharyngeal polypi springing from the submucous periosteum.

5. *Cancer.*—We have already remarked that cancer not unfrequently forms the basis of exuberant epithelial formations and erectile tissues. It further occurs in the larynx as fibrous cancer, in the submucous areolar tissue as a medullary cancer, and (which is extremely singular) as cancerous degeneration of the *arytenoid cartilages.* According to the volume of the morbid product, there are found larger or smaller nodular roundish protuberances into the laryngeal cavity, which thus becomes more or less diminished in size. These cancerous tumors, for the most part,

prove fatal while still in their state of crudity; they sometimes, however, undergo their ordinary course of metamorphosis, and give rise to a cancerous ulcer.

Cancer also occurs in the trachea and bronchi, but in such cases it is almost invariably only a secondary affection. Thus it occasionally happens that the trachea is perforated by surrounding masses of medullary cancer in the neck; more frequently, in which case, the bronchi may also be affected, by cancerous accumulations in the mediastinum posticum, or by cancer of the œsophagus; and in these cases the air-passages become contracted by the growth of vegetations within them. In the bronchi we sometimes observe an ordinary cancerous degeneration of the fibrous sheath, proceeding in different directions from a bronchial stem along its ramifications, by which their walls are thickened and rendered rigid, and their calibre is diminished, while their inner surface becomes nodular and uneven. This degeneration appears to arise from cancerous disease of one or more of the bronchial glands.

6. *Tuberculosis of the Air-Passages.*—Tubercle is very commonly met with in the air passages, but it is found in some parts of them much more frequently than in others. The most common position is in the the larynx; it is very rare in the trachea and larger bronchi; while, again, it not uncommon in the ultimate ramifications of the tubes. On softening it gives rise to tuberculous ulceration, and to laryngeal, tracheal, or bronchial phthisis, according to the seat of the deposit.

Tuberculosis of the larynx, as a primary and independent affection, is so extremely rare that we feel inclined to doubt its existence. It is almost invariably developed as a consequence of pulmonary tuberculosis, and then, as a general rule, not until the affection has established itself as a pulmonary phthisis, and made considerable progress. The seat of tubercle is almost constantly and exclusively the mucous membrane and submucous areolar tissue lying over the transverse muscle and the adjacent arytenoid cartilages; it occurs, however, exceptionally at other spots, as for instance, the anterior surface of the epiglottis. It is either deposited in the form of gray granulations in the submucous areolar tissue, or as yellow, caseous, friable, tuberculous matter, is infiltrated into the mucous membrane; in either case, however, and especially in the latter, it rapidly softens, and ulceration is established. The softened, gray granulations form small roundish ulcers, varying from the size of a millet-seed to that of a lentil, with raised, hard edges. These unite with one another, and give rise to a secondary form of ulcer, irregular in shape, with pouch-like indented edges, and a cellular, callous, thickened base, both of which may become the seat of secondary tuberculous deposit. The tuberculous infiltration becomes disintegrated in the mucous membrane, and forms therewith an extremely irregular and, as it were, gnawed and fissured ulceration, presenting obvious signs of reaction, namely, redness, injection, swelling, œdema of the tissues, and aphthous exudation over the adjacent parts.

The ulcer enlarges in consequence of secondary tuberculous deposition at its edges and the surrounding parts, as well as on its base, superficially as well as deeply; it thus gives rise to ulcerations which extend over the whole larynx and epiglottis, upwards to the soft palate and root of the tongue, downwards to the trachea, and inwards, causing suppuration and necrosis of the fibrous tissues and of the cartilages. They may even perforate the larynx from within outwards, and give rise to emphysema.

The secondary tuberculous ulcer is sometimes distinguishable by a condylomatous development of the mucous membrane at its edges, and of the islets of mucous membrane, which, as the ulcer enlarges, are frequently left on its base. It is not improbable that, in these cases, the tuberculosis is combined with syphilis.

There can be no doubt that in some rare cases, and under the requisite general conditions, tuberculous ulcers of the larynx are healed; but they always leave an unshapely cicatrix, puckered in proportion to the extent of the ulcer, and callous in proportion to its depth. We must, however, be careful not to regard all the cicatrices which we may find in the neighborhood of true tuberculous ulcers in the larynx and trachea, as the cicatrices of so many tuberculous ulcers.

Tuberculosis of the Trachea is extremely rare, it being only seldom that the corresponding laryngeal affection extends itself in a tuberculous ulcer to the upper part of the canal. In laryngeal phthisis we often, however, meet with small ulcers on the tracheal mucous membrane, and frequently in such numbers that they present a confluent appearance. These are the ulcers to whose cicatrices we referred, as liable to be mistaken for the cicatrices of tuberculous ulcers. They are small, shallow ulcers, most commonly of an oval but sometimes of a linear form, with a very slightly concave base; the depression being so trifling that it is sometimes only detectible when the light falls obliquely on it: this base presents a raw and excoriated appearance, is of a pale or dark red color, and is either exposed or coated with a creamy, diffluent exudation of a croupous nature; and it is surrounded by a fiery redness, or by a sharply defined red areola. They are most commonly situated on the posterior wall of the trachea, and frequently extend into the bronchial trunks, and we often find them much more numerous on one side (that, namely, in which the most diseased lung is situate) than on the other; moreover they are found with tolerable frequency in the pharynx and on the mucous membrane lining the mouth. They present nothing in common with tuberculous ulcers, and consist, as is at once seen, in an exudative aphthous process which is frequently associated with florid, laryngeal phthisis. If the tuberculosis be in a state of arrest, or actually retrograding, the above-described erosions become replaced by delicate, whitish, glistening, radiating, or star-like cicatrices.

Bronchial Tuberculosis.—This is seated in the bronchial mucous membrane, which becomes so infiltrated with yellow, lardaceous caseous, tuberculous matter, as finally it appears converted into it. The bronchial tube itself becomes considerably enlarged, its calibre becoming at length completely obstructed by tuberculous matter, while its fibrous sheath becomes infiltrated with lardaceous matter, callous, and thickened. This degeneration sometimes attacks the bronchial mucous membrane as a *secondary* affection, in which case it arises from tuberculous abscesses, and affects the tubes opening into them; it is then primarily dependent on pulmonary phthisis.

Primary bronchial tuberculosis is a much more important affection. It is, as we have already mentioned, a disease of the ultimate ramifications of the bronchial tubes, arising originally in them, and extending backwards to the larger bronchi. Like pulmonary tuberculosis, it most commonly occurs in the bronchial ramifications of the upper lobes, but it stands contrasted with that affection in frequently occurring in the peripheral branches; it attacks a large portion of the bronchial tree, and on making a section of the pulmonary parenchyma we find it traversed by thick-walled, dilated, bronchial tubes, filled with caseous tuberculous matter.

Bronchial tuberculosis, although very frequently combined with lardaceo-gelatinous, or fatty and caseous tuberculous infiltration of the lungs, sometimes occurs as an independent disease. In the latter case the obstruction in the bronchial tubes gives rise to obliteration of the pulmonary vesicles and obsolescence of the parenchyma connected with them; and, on making an incision, we then find the obstructed tuberculous bronchi ramifying through the parenchyma in the form of ribands of puckered, tough, elastic tissue.

The tuberculous matter may undergo either of the two following changes. It may soften; and in this way it not unfrequently destroys the bronchial walls, and gives rise to tuberculous abscesses in the adjacent parenchyma. The abscesses arising primarily from the destruction of the bronchus are incomparably rarer than those arising from the softening of pulmonary tubercles. This metamorphosis is most likely to occur when tuberculous infiltration of the parenchyma is simultaneously present. The other change to which we referred is the cretefaction of the tuberculous matter. This metamorphosis most commonly occurs when the bronchial tube has been completely obstructed by tuberculous matter, and the pulmonary tissue to which it pertains has become obsolete; under favorable conditions it seems, however, sometimes to occur in other cases, in which the morbid product becomes as it were disintegrated into a caseous pultaceous mass, which, instead of becoming softer thickens and becomes ultimately converted into a chalky substance, around which the bronchial tube becomes contracted and atrophied.

Bronchial tuberculosis, as a primary affection, is most common in childhood, and is usually associated with all the tuberculoses of other organs peculiar to this period of life, and especially with intense *tuberculosis of the bronchial glands.*

Its most marked analogies are exhibited in tuberculoses of the mucous membrane of the Fallopian tubes and uterus.

§ 7. *Anomalies of the Contents of the Air-Passages.*—We must here notice:

1. The products of various morbid processes on the mucous membrane of any portion of the air-passages, such as blood, or as mucus (which may collect in large quantity, and may present many peculiarities of character, being gray or pearl-colored, colorless, transparent, aqueous, viscid, glassy, of a creamy, whitish-yellow color, or puriform), or as true pus, or membranous exudations (croup-membranes), or as ichorous fluid, tubercle and tuberculous pus, fragments of necrosed cartilage, ossified bronchial cartilage, &c.

2. Products of morbid processes, occurring external to the air-passages, and finding their way into them, either by the natural passages, or by a destruction of tissue,—as for instance *blood* (usually in considerable quantity), in a coagulated or fluid state from the lungs or from aneurisms which have opened into the air-passages; *serous frothy fluid* (bronchial froth) from the parenchyma of the lungs; *pus and ichor* arising most frequently from abscesses in the lungs and bronchial glands or in the vertebræ; the corrosion of a bronchial trunk may allow the fluid of empyema to be discharged into the air-passages, and by another process the contents of an hepatic abscess may make their way into the bronchial tubes: the *ichor of cancer*, principally arising from cancerous destruction of the œsophagus; *masses of tubercle; calcareous* and *stony concretions*, with which, as produced in the air-passages, we must place cretified blennorrhœal mucus and tubercle; and, finally, *acephalocysts* from the lungs, liver, and thyroid gland (Portal).

3. Foreign bodies which—

(*a.*) Find their way from the pharynx and œsophagus, or even from the stomach and intestinal canal, by means of abnormal modes of communication, or by the natural passages, into the air-tubes. The most common are fluids that have been imbibed, and having made their way through ulcerous or cicatrized strictures, penetrate into the trachea or bronchi. Amongst the foreign bodies proceeding from the stomach and intestinal canal, we must especially notice the round worm, which has been seen both by ancient and modern observers in the pharynx in children, and, crawling into the glottis, has produced death by suffocation.

(*b.*) Articles of food may become impacted in the glottis, in cases in which the act of deglutition is impeded by inflammation, and degeneration of the muscular walls of the pharynx, or by enlarged tonsils, or is inattentively performed from haste and carelessness as in imbeciles, or is interrupted by coughing, sneezing, or laughing. These accidents are most liable to occur when, from atrophy, rigidity, or malformation, the epiglottis is no longer able sufficiently to protect the glottis; and the substances most commonly causing them are large pieces of tough meat, skin, and gristle.

(*c.*) Foreign bodies which accidentally enter the glottis independently of any intentional act of swelling, and either fall into the trachea and bronchi, or are forced into them by an automatic movement of deglutition. Cases are recorded in which the following bodies have got in the air-passages:—small arrows, plum and cherry stones, small coins, natural and artificial teeth, grains of corn, nails, pebbles, and fragments of glass. In favorable cases they are soon removed by coughing; but otherwise they remain for a long time in the air-passages, inducing not only inflammation of the mucous membrane, pneumonia, and ultimately suppuration, but even destruction of the walls of the bronchial tubes. This they may effect in various ways, and they may even penetrate into the adjacent bloodvessels. I may here mention the following singular case: A little boy sucked a dart from a blowing-tube. Its feathered portion was downwards as it descended the trachea, and from thence it went into the left bronchus. On the twelfth day he died from hemorrhage of the air-passages, having at the same time symptoms of pneumonia. Dissection revealed bronchitis, especially on the left side, and hepatization of the left lower lobe. The dart was lying loose in the left bronchus, with its feathered part downwards. Opposite to the opening of this bronchus into the trachea, in the cartilaginous wall of its right side, there was an injured spot of about the size of a hemp-seed, and through this there was a perforation into the adjacent arteria innominata. In the paroxysms of coughing, the point of the dart was being constantly forced against this spot in the right tracheal wall, which lay opposite to the axis of the left bronchus; and in this way the fatal lesion was ultimately produced.

It is neither unimportant nor uninteresting to remark that in the majority of cases, these foreign bodies fall into the right bronchus (Key). This is undoubtedly dependent on its larger size, on the greater obtuseness of the angle that it makes with the trachea, and on the greater energy of the current of air rushing through it. Moreover this is in accordance with the well-known fact that in new-born children respiration is effected sooner and more perfectly by the right bronchus and lung than by the left.

II.—ABNORMAL CONDITIONS OF THE PLEURA.

§ 1. *Deficiency and Excess of Formation.*

The pleural sacs are altogether absent when there is an entire deficiency of the respiratory apparatus, as in cases of acephalia, the thoracic cavity being then filled with a dense fibro-cellular tissue. A partial deficiency occurs when, in consequence of the absence of the diaphragm, the pleura and peritoneum form one continuous membrane.

An excessive development occurs in the form of *duplication* of the pleura, except in cases of double monstrosity, when the thoracic cavity is more or less doubled, or the two lungs are deposited in a large common cavity. It is, however, extremely rare, especially as contrasted with the frequent occurrence of congenital duplications of the peritoneum; and on this account, as well as in consequence of its relations, I would direct attention to a hitherto unnoticed persistent duplication of the *right* pleural sac. It forms, at the obtuse apex of the pleura, a fold, hanging from above downwards, and from without inwards, including the arch of the vena azygos, and lying in a supernumerary fissure, which divides the upper lobe into two parts.

§ 2. *Anomalies in the Size and Form of the Pleural Sacs.*

The size and volume of the pleural sacs are proportional to the congenital or acquired volume of the lungs; thus, for large lungs there are wide and spacious pleuræ, while for small lungs the pleural sacs are less capacious. When the lungs become enlarged, as in emphysema, they also enlarge; when the lungs from any morbid process, become atrophied, they also diminish in a corresponding degree. Moreover the pleural cavities may undergo various degrees of enlargement by the accumulation within them of gases or liquids, and may be diminished in various ways by an increased size of the peritoneum or pericardium, by adventitious products in the mediastina, or by malformation of the bony case of the thorax.

The form of the pleural sacs is regulated by that of the osseous thorax, and we must refer our readers to the remarks on the anomalies in the form of that cavity.

§ 3. *Diseases of Texture.*

a. Hyperæmia of the Pleura.—A continuous increased flow of blood to this membrane gives rise, according to its extent, either to local or general opacity, and to hypertrophy; and ultimately to the development of an anomalous cartilaginous and osseous substance in its texture, and in that of the subjacent areolar tissue.

On the other hand, congestion may cause increased secretion, which, in accordance with its character and the general state of the blood, may terminate in a temporary or permanent accumulation of various quantities and kinds of serous fluid, constituting hydropspleuræ or hydrothorax. It is very seldom that it leads to hemorrhage of the pleura or hæmothorax.

b. Inflammation of the Pleura (Pleuritis, Pleurisia).—This is the most frequent of the diseases of the pleura; it generally appears as an idiopathic and primary affection, most commonly of a rheumatic nature; in consequence of a wound or shock affecting the walls of the thorax, or of the contact of the pleura with the atmospheric air, either from without or through the air-passages from within, or of the contact of pus, ichor, &c.; or it may originate in the extension of inflammation or other morbid processes in neighboring parts, and especially the lungs; and it very often is a secondary or metastatic process, and is then frequently associated with inflammation of other serous membranes, especially of the peritoneum and pericardium; it generally exhibits a very well-marked croupous character.

Pleurisy is either *general* or *partial and circumscribed:* in the former case the process on the visceral surface (the pulmonary pleura) as a general rule exhibits comparatively little intensity. In either case the disease may be *acute* or *chronic.* As everything that has been stated with respect to inflammations of serous membranes generally applies to inflammations of the pleura, we shall here limit our remarks to the notice of certain important peculiarities presented by this membrane.

The exudations present all the differences that we have there described; but here we must especially notice, as frequent and very important forms, the *purulent* exudation (empyema) and the *hemorrhagic.* In relation to the quantity of the exudation, acute, and more especially chronic pleurisies depositing a long-continuing and paroxysmally increasing exudation in large quantity, are of the greatest importance. When the pleurisy is general, the exudation sometimes amounts to eight, ten, sixteen, or twenty pounds, and occasionally even more. The walls of the thorax become in these cases more or less dilated; the intercostal spaces are enlarged, and in consequence of the paralyzed state of their muscles are flattened; the diaphragm is forced downwards in the abdomen; the mediastinum and the heart are pushed to the opposite side, and thus diminish its capacity. The lung itself is compressed by the effusion, in a degree corresponding to its quantity, and if no old adhesion exists to oppose it, it is constantly pressed upwards and inwards on the mediastinum and the vertebral column. We find it thus compressed to the fourth, sixth, or even the eighth part of its normal volume, and so flattened on its external arched surface as to present the appearance of a flat cake; its texture is of a pale red, bluish-brown, or lead-gray tint; and it is of a leathery toughness, and devoid of blood and air. In this state its external surface is invested with a plastic coagulum; as this extends over it to the costal pleura, the lung is, strictly speaking, excluded from the cavity of the sac formed by the pleuritic exudation. If adhesions already exist, as the remains and consequences of previous inflammations, they will, in proportion to their position, distribution, the tissues comprising them, and their powers of resistance, present a certain amount of opposition, as we have already described, to the displacement; and the degree of dislocation of the lung must be thereby more or less modified. In partial pleurisy, the displacement and compression are limited to the portion of the lung corresponding to the extent of the affection.

The *purulent* exudation most commonly accompanies the pleuritic process in weak, cachectic persons, whose organisms seem prone to form pus; on the other hand, it may, by an intensely high degree of inflammation, and by its frequent recrudescence, give rise to very rapid general debility, cachexia, and pyæmia. The effused pus not unfrequently degenerates into ichor, and this change is sometimes accompanied by the development of gas, so that from its decomposition and disintegration a pneumothorax becomes added to the purulent effusion. This not unfrequently leads to suppuration of the walls of the chest, with or without caries, and to the spontaneous discharge of the collected fluid, or to suppuration of the lung from its surface, leading to perforation of the bronchial tubes, or to suppuration of the principal bronchial trunks, whereby, on the one hand, atmospheric air is allowed to penetrate the pleural sac, and, on the other, pus enters and is discharged through the bronchi.

Amongst *partial* pleurisies we must especially notice those which occur about the apices of the lungs (for the most part dependent on pulmonary tuberculosis), those in the inferior portion of the pleural sac and on the pleura diaphragmatica, those on the laminæ of the mediastinum, and finally those which occur in the interlobular fissures of the lungs.

Plastic exudations, when they result either from an acute or a chronic simple process (by which we mean to imply a process not implicating the exudation), become transformed into areolar tissue of various degrees of density, into areolo-serous and fibrous tissues, presenting varied relations in reference to form and distribution.

When they are the products of general pleurisy they invest the whole of the costal and pulmonary pleura; or they merely occur at individual spots in the form of circumscribed patches on the serous membrane.

The areolar tissue forms either dense or rigid bands, or thready, lax, and movable adhesions, according to comparative absence or presence of aqueous effusions, occurring either originally or during its formation. These adhesions are of very common occurrence; when the lung adheres at every point of the parietal surface of the pleura, they are termed *general*, and when the latter is only attached to a portion of the lung, *partial* (*or cellular*) *adhesions*. They may undoubtedly become the seat of new inflammatory processes, but, as Laennec remarked, they usually limit the progress of new pleurisies. When general dropsy and the dropsical diathesis are present, they may become the seat of a serous, briny infiltration.

Sometimes there are no adhesions, the tissue forming delicate flocks or cumuli, which are seen scattered over both surfaces, and not on corresponding points; in many cases delicate areolar adhesions appear to have given way from tension and friction, the patches of new tissue being then on corresponding spots on the two pleural surfaces; these are usually either conical with a broad base, or are drawn out into string-like prolongations.

If, during the organization of the exudation, an aqueous effusion were present, which prevented the contact of the two lamellæ, then the areolar tissue receives a serous investment, and the pleura becomes clothed with a second, and newly-formed, tolerably easily removable serous membrane.

Where the exudation is thicker, it becomes converted under similar conditions into a smooth, bluish-white fibrous lamella, which either invests the whole pleura, or adheres to it at particular spots, which are either clearly defined at their circumference, or gradually and imperceptibly merge into the surrounding tissue. Even in the first case this lamella presents no general uniformity of thickness, and exhibits an areolar, cribriform, broken appearance; and then it sometimes happens that after the diminution of the aqueous effusion, the lung becomes more strongly developed at those points where the false membrane is thinnest, and bulges through the meshes in the form of mammillary appendages.

Chronic inflammation, when it insidiously extends itself amongst the organizing coagula, occasions very important metamorphoses of the substance of the pleura, and of the products that are evolved. It very frequently gives rise to hemorrhagic or serous effusions, in which there are found coagula of considerable thickness, which become gradually converted into very dense and resistant fibrous bands. As the result of general pleurisy, we have not only dilatation of the thorax corresponding to the amount of effused fluid, and the already mentioned displacement and compression of the lung, but there are likewise formed thick, pseudo-membranous pleural sacs, adhering to both surfaces. The parietal wall is usually by far the thicker, not unfrequently measuring four, six, eight, or even ten lines, and occasionally even an inch in thickness.

If, under these conditions, the serous effusion becomes diminished by absorption, the lung, invested by a fibrous pseudo-membrane, may either again very slowly attain to a certain degree of development, or it may remain unalterable, according to the thickness and resistance of the membrane; in the former case the walls of the thorax approximate for the purpose of filling the vacant space, till finally, when the serous effusion is thoroughly removed, the two lamellæ of the fibrous exudation come in contact, and coalesce with each other. Hence, as was shown by Laennec, the thorax becomes permanently contracted, the amount of the contraction varying from a scarcely perceptible flattening to a very obvious pitlike depression, modifying the form of the chest. In these latter cases the greatest depression usually occurs in the neighborhood of the 6th, 7th, and 8th ribs, and the lateral wall of the chest presents a well-marked concavity from the axilla downwards over the ribs. The thorax, if measured, is found contracted not only in its circumference, but in all its diameters. The depression is most marked where the ribs are the most sunk, and the latter sometimes even touch and press upon one another. The surrounding muscles are emaciated, especially the intercostals, which become shrivelled in proportion to the degree and continuance of the paralysis they have undergone, and are finally converted into fibro-areolar tissue. The dorsal portion of the spinal column gradually deviates with a lateral curvature towards the healthy side, and the shoulder of the affected side sinks proportionally deeper. In the lumbar region there is a curvature corresponding in degree to that in the dorsal region, but inclining to the opposite direction, giving to the pelvis a higher position on this side, and causing an apparent shortening of the corresponding lower extremity. In this way the form of the hips and buttocks, and the general bearing or carriage of the body, present a certain similarity with the corresponding appearances after coxalgia.

There are several causes for the contraction of the thorax after these forms of pleurisy. Amongst these we may especially mention—(1), the pressure of the atmosphere on the thorax, when it is impossible for the pulmonary parenchyma to regain its former condition with a rapidity equivalent to that with which the serous effusion is absorbed, in consequence of the fibrous sheath investing the lung, and the destruction of its extensibility and elasticity by continued pressure;—(2), the tendency exhibited by the costal lamina of the fibrous exudation to increase in density and to contract; and (3), the similar tendency on the part of the fibro-areolar tissue that has taken the place of the atrophied muscular tissue in the intercostal spaces.

Contraction, even when arising from general pleurisy, may affect only one portion of the chest; the upper portion may be thus modified, while the lower remains either absolutely dilated, or, at all events relatively so, as contrasted with the sunken portion. Thus, after partial resorption of the effused fluid, an adhesion of the lamellæ of the fibrous exudation may take place, while inferiorly they are separated from one another by the effusion stagnating between them. Partial contractions of the thorax are most commonly the result of partial and circumscribed pleurisies, as we see from the depression of the thorax in the neighborhood of the clavicles, in consequence of pleurisy being associated with pulmonary tuberculosis around the apices of the lungs; and from the contraction of the lower part of the thorax, in consequence of pleurisies about their base.

The fibrous exudation, and especially its parietal lamella, sometimes undergoes ossification; this change occasionally occurs before, but more generally after, the complete absorption of the serous effusion. The deposition of osseous matter, for the most part, occurs in the thickest portions of the exudation in the form of compact nodulated strings and plates. There are rare cases in which thin osseous plates are formed over the whole pseudo-membranous pleural cone, excepting the thin layer investing the lung, and if this change should occur before the complete absorption of the effusion, this will remain permanently enclosed in an osseous sac.

As some of the above-named causes of thoracic contraction are equally present in pleurisies accompanied by other forms of exudation, so we find these contractions in and after their cure, although, generally speaking, they are not so distinctly marked. Thus we observe a slight contraction when the visceral lamina of the exudation has become organized into extensible and yielding areolar tissue, and when, notwithstanding that the lung has regained its original size, the costal lamina has been converted into a thick fibrous sheath; in this case the contraction is consequent on the condensation and shrinking of this lamella. Moreover, after pleurisies with an inconsiderable amount of plastic coagula, and with purulent exudation, we observe thoracic contraction; this is then produced, on the one hand, by paralysis and atrophy of the lung, caused by the long-continued pressure of a large quantity of exudation, and on the other, by the paralysis and consequent histological alteration of the intercostal muscles, which keep equal pace with the intensity and duration of the inflammatory process, and with the quantity and coagulation of the exudation.

Pleurisy sometimes occurs simultaneously on both sides; in these cases both sides may be attacked at the same time, or the pleurisy on one side may succeed that of the other.

Pleurisies with long-standing effusion may give rise to cachexia, general dropsy, hydrothorax on the opposite side, hyperæmia and œdema of the lung of the same side, asphyxia, dilatation of the right side of the heart, venosity, more or less obliteration of the lung, and thus not unfrequently to the eradication of a pre-existing tuberculosis.

There is a *typhous* pleurisy, which, in the strict limitation of the term, is almost always associated with typhous pneumonia.

We have already mentioned, that one of the terminations of empyema consists in suppuration of the pleura; but *this*, and *a similar destruction*, may sometimes arise from the pulmonary parenchyma itself, as well as from without it; in the latter case, abscesses lying on the surface of the thorax, accumulations of tuberculous pus in the sternum and its vicinity, or in the vertebral column, softening encysted exudations on the peritoneum diaphragmaticum, perforating abscess of the liver and mammary gland, ichorous cancer, &c., may be regarded as exciting causes. Perforation of the costal pleura occurs for the most part where there is a tolerably thick and resistant layer of exudation deposited by a previous pleurisy, and adhesion thus established with the lung; there is then no discharge of pus into the thoracic cavity, but it not unfrequently happens that it finds its way through fistulous passages into the adherent lung, and leads to its ulcerous destruction.

1. *Gangrene of the Pleura* occurs in consequence of its being denuded by accumulations of pus or ichor in the costal or pulmonary wall. The pleura then assumes the appearance of a yellowish-white, or more frequently of a blackish or greenish-brown, lax or deliquescent slough, with superficial gangrene of the lung. There is no difficulty in distinguishing it from the acute black softenings to which the pleura diaphragmatica is

subject from the stomach, or the left layer of the mediastinum from the œsophagus.

c. *Adventitious Products.*—Passing over the areolar and areolo-serous adventitious products, we must notice *anomalous fibrous* and *cartilaginous tissues*, and *anomalous osseous substance.* These are especially frequent in the pleura, and in relation to their origin and their seat they present two distinct varieties. *In one case* they are products of inflammation, which, as has been already stated, become converted into fibroid and cartilaginous tissues, and then into concretions of bone-earth in the form of strings and plates; these are seated on the inside of the pleura, with which they intimately coalesce. *In the other case*, the fibroid and cartilaginous tissues are developed independently of inflammation, and merely in consequence of a hyperæmic condition in the subserous areolar and fibrous tissues, and in the tissue of the serous membrane itself. We first observe a whitish, more or less circumscribed opacity and condensation of the serous tissue; there is here a development of tissue resulting in the formation of a smooth or nodular elastic plate, or of a group of granulations of fibrous or fibro-cartilaginous tissue, or even of irregularly shaped masses, which vary from the size of a pea to that of a nut, and finally ossify. These are always situated under or on the outer side of the serous membrane, and are invested by it.

These two varieties may be easily distinguished from one another: fibrous exudations invest the costal as well as the pulmonary pleura; they ossify, however, only on the costal pleura. Subserous adventitious products occur almost exclusively on the costal and diaphragmatic pleura, and their most common seat is in the intercostal spaces. They sometimes become liberated, and are found free in the cavity of the thorax, in the form of round, nodular masses of cartilage.

2. *Tuberculosis of the Pleura.*—Pleural tubercle occurs in all the forms in which we have stated that tuberculosis attacks serous membranes; that is to say—

(*a*), as a rapid metamorphosis (either complete or partial) of a pleuritic coagulum; this is most common on the parietal lamina;—

(*b*), as tuberculous formation in a pseudo-membranous coagulum that is either becoming organized in some manner or other, or that has become organized to a certain stage; as, for instance, tubercle on and in layers of areolar, fibro-areolar, and other exudations;—

(*c*), as primary acute tuberculosis in the form of the most minute miliary tubercles.

The second kind of tuberculous formation is very frequently associated with secondary inflammation of the pseudo-membrane serving as the base to the tubercle, and in these cases we may frequently observe hemorrhagic exudation.

Pleural tubercle is, in most cases, the result of a dyscrasia that has already revealed itself in tuberculosis of a parenchymatous tissue, most probably of the lungs or bronchial glands; sometimes, however, it occurs as the first in the series of successively developed tuberculosis, and not unfrequently it is associated with quiescent or even retrograde pulmonary tuberculosis, indicating a tumultuous recrudescence of the general disease, and from that period being frequently combined with general tuberculosis.

Pleural tubercle not unfrequently softens and gives rise to tuberculous abscesses in the different pseudo-membranous structures in which it is deposited. These abscesses may penetrate the pleura, and even the thoracic walls, with or without caries.

3. *Cancer of the Pleura.*—Cancer of the pleura is much rarer than tubercle. It never occurs as the first in the series of cancerous deposits occurring in an individual, but is always the result of a cancerous dyscrasia that has previously developed itself in other structures. It is very frequently associated with cancer of the mammary gland, with cancer in the mediastina, with cancer of the osseous system, or with general cancerous disease; it is commonly developed simultaneously with cancer of the lungs, and usually runs a very rapid course after the extirpation of large masses of cancer.

The pleura may be perforated from without by adjacent cancerous formations which, after having established the cancerous metamorphosis on it, sprout forth on its cavity in the form of nodular growths; or the cancer may appear primarily on its inner smooth surface, in the form of flattened, roundish or nodular, lardaceous or medullary masses, varying from the size of a hemp-seed to that of a hen's egg, or even of a man's fist. They are sometimes so fluid as to be diffused and form a stratum, and may be either loosely or firmly attached to the serous membrane on which they are situated. In the latter case they involve the tissues of the pleura itself, and in general turn out to be *medullary cancer.*

Their presence always occasions an accumulation of more or less serous fluid in the pleural sacs.

§ 4. *Morbid Contents of the Pleural Sacs.*—In addition to the anomalous contents of the pleural sacs, which have been already mentioned, and to which we shall have occasion subsequently to allude, we shall only at present treat of the presence of gas and serum in the pleural sacs (*pneumothorax* and *hydrothorax*).

There are many sources from which gases of various kinds may accumulate in the cavity of the thorax, and (independently of the atmospheric air finding its way there from without, through a penetrating wound in the walls of the chest, or through the bronchi in injury of the lung with or without an opening in the thorax, or through both causes combined), a pneumothorax may occur under the following conditions:

(*a.*) In consequence of the opening of a tuberculous abscess before there is perfect adhesion of the lung to the walls of the chest through pleuritic exudation. Here we must particularly direct attention to the abscesses which are preceded by superficial tuberculous infiltration arising from softening.

(*b.*) In consequence of the softening and separation of a superficial gangrenous eschar of the lungs.

(*c.*) In consequence of the softening of the so-called metastatic deposits which penetrate the pulmonary pleura before reactive hepatization can occur.

(*d.*) In consequence of the opening of a pulmonary abscess communicating with some of the bronchial tubes.

(*e.*) From the development of gas from the decomposition of purulent and ichorous exudations (empyema).

) In rare cases, a certain quantity of air is found in the pleural sac, product of the inflammatory process, and associated with a benign ·itic effusion.

.) In cases in which there is an opening formed into one of the chial trunks, in consequence of its corrosion by purulent and ichorous ation.

.) In consequence of the laceration of one or more of the superficial onary vesicles in vesicular emphysema, or from rupture of the ·a in interlobular and sub-pleural emphysema.

) From perforation of the diaphragm or of the mediastinum, aris- rom acute softening of the stomach or œsophagus.

hen the disease giving rise to the pneumothorax is not in itself fatal. h, however, is the case in softening of the stomach and œsophagus, the gas accumulated in the thorax invariably causes inflammation e pleura, and exudation, compression of the lungs, dilatation of the ıx, &c.; the nature of the exudation varying according to the source, re, and products of the gas, and of the other substances which are ltaneously extravasated into the pleural sac.

ydrothorax is a very rare disease, if we except those cases in which nstitutes a part of general dropsy; and even then, if we exclude in which it is dependent on cardiac or pulmonary diseases, it is rarely the first in order of the various serous effusions. It may as a consequence of hyperæmia of the pleura, and in that case the ing and hypertrophy of the serous membrane are proportional to luration of the effusion. It is also often present when there are erous vegetations on the pleura: but its most common exciting causes diseases of the heart and large vessels, pericarditis, catarrh and chial dilatation, indurated hepatization, and pleurisy; finally it s a portion of the general dropsy consequent on diseases of an ex- tive nature, such as typhus and puerperal fever, emaciation from ·cles or cancer, and Bright's disease of the kidney.

hen there is a large quantity of air in the pleura, the lung becomes aced and compressed in the same manner as in pleuritic effusions.

should be most carefully distinguished from the serous effusion h occurs as one of the processes of inflammation of the pleura. The erienced observer may find some difficulty in the establishment of a ct diagnosis between it and those pleurisies which deposit exudations ient in plastic matter (the so-called active dropsies), especially when latter have existed for a considerable period.

III. ABNORMAL CONDITIONS OF THE LUNGS.

l. *Deficiency and Excess of Formation.*

very imperfect monsters, as for instance, in cases of acephalia, the as well as the central organs of circulation, are altogether absent. wer degrees of monstrosity, and even in cases in which, in other cts, the organization is normal, there may occur a considerable defi- y of one or both lungs, that is to say, an arrest of development. development may be arrested at so early a stage, that we can barely ve them as roundish little bodies seated on the bronchi. It generally happens that this condition is a consequence of the contraction of the volume of the thorax, or of pressure exerted on the lungs either by the displacement of the abdominal viscera into the thorax, in cases of absence of the diaphragm, or by the accumulation of fluid in the thorax.

An excessive formation occurs in double monsters, as more or less duplication of the lungs, either with or without a simultaneous duplication of the pleural sacs. The occurrence of an extraordinary number of lobes is apparently due to an excess of development.

§ 2. *Anomalies of Size.—Hypertrophy and Atrophy.*

The manifest differences in the size of the lungs are, for the most part, dependent on the number and the capacity of the air-cells. As an individual structure, the lung will attain a large size when there is a large number of air-cells, and at the same time their volume is well developed; while under opposite circumstances, the lung will appear small.

The former usually occurs in the male sex, and is generally associated with well-developed muscular and osseous systems, and with a low state of development of the abdominal viscera, whilst the latter is most common in the female sex, and is associated with a weak state of the muscular fibre, fragility of the bones, and a preponderating development of the abdominal viscera.

The lungs may appear large either within the normal limits or beyond them, when a certain number of the air-cells are dilated; and under opposite conditions they may appear small or abnormally diminished; indeed, the former may occur when the number of dilated cells is comparatively small, and the latter when the number is comparatively great. In the former condition the pulmonary tissue is rarefied, in the latter it is compressed and dense.

Hence in forming an opinion of the size of the pulmonary organs in any individual case, the density of the parenchyma should be especially noted, since it alone determines the respiratory capacity of the lungs; and the two extremes, excessive rarefaction and excessive condensation, constitute very important morbid conditions, of which we shall speak in our observations on changes of tissue.

Hypertrophy of the lungs undoubtedly arises from a remarkable combination of dilatation of the air-cells, with a simultaneous augmentation of their tissues; we sometimes observe it as a vicarious development of one lung, when the other, for any reason, is no longer able to discharge its proper function. It is incontestable that this hypertrophy does not consist in an increase in the number of the air-cells, but in their dilatation, in the increased thickness of their walls, in the enlarged calibre of the capillaries, and in the development of new vessels. The pulmonary tissue is thus rendered denser and more resistant, and the lung exhibits a singular power of resisting external atmospheric pressure; it has become *larger*, and its thoracic cavity wider.

Atrophy of the lungs is the exact reverse of this condition. It is most marked in advanced age, when it is known as *atrophia senilis;* sometimes, however, it occurs in the earlier periods of life, constituting a premature involution of the respiratory organs, and it is only in these latter cases that it strictly falls within the domain of pathological inquiry. It consists in a dilatation of the air-cells (emphysema), and an alteration from their angular or polygonal into a round or elliptic form. The dilatation is the consequence of the emaciation and attenuation of their walls, the vessels of which become obliterated. The atrophy of the cell-walls may proceed to such an extent that several cells may coalesce; the interlobular areolar structure has then disappeared, and hence the lobular structure no longer exists; the pulmonary tissue then represents an irregular, perforated network; the lung itself is of a pale, light-gray color, generally dotted with numerous specks of black matter, conveys a soft downy sensation to the hand, is light and small, and collapses on the opening of the thorax; on cutting into it the air escapes tardily, with a dull and diffused sound. Its tissue is dry and bloodless.

Thus marasmus of the pulmonary organs is, for the most part, associated with an equally well-marked atrophy and dilatation of the trachea, emaciation of its walls and deficient moisture of its mucous membrane, the nature of the changes being in both organs the same. Moreover, as a general rule, it is most highly developed in the superficial portion of the upper lobes, giving rise to a singular displacement of the interlobular fissure, and causing it gradually to assume a vertical position (Hourmann).

The thorax becomes depressed over the atrophied lungs, exhibits distinct lateral flattening, and assumes a conical form; there is an arched curvature of the vertebral column in a backward direction; the sternum is thrust forward, and there is a diminution in the vertical diameter in consequence of the curvature of the spine, and the absorption of the intervertebral cartilages, and partially even of the vertebræ themselves. The soft parts about the thorax disappear; the muscles become emaciated and pale; the diaphragm thin, lax, and plicated; and the heart small.

On these changes are based the difficult respiration, and, in a great measure, the collapse, pallor, and lividity of the tissues,—in short, the general atrophy of old age. The impaired state of the respiratory muscles renders the act of inspiration difficult and imperfect; the deficient contractility of the pulmonary tissue, together with the above-named muscular weakness, opposes similar obstacles to the act of expiration; while the surface of the lungs presents to the atmospheric air so obliterated a capillary network, that only a small quantity of blood can be submitted to the vivifying process of arterialization.

If this change occur at an earlier period of life, and an abnormal relation be then established between the degree of involution of the lungs and that of the other organs concerned in hæmatosis, the disease will assume much more importance if active dilatation of the right side of the heart should supervene.

A remarkable enlargement of the lungs takes place in emphysema; a less marked and usually only partial enlargement also occurs in hepatization, in highly advanced tuberculosis, in cancer of the lungs, &c.

Diminution of the lungs is chiefly induced by contraction of the thorax, the pressure of gases or liquids that have accumulated in the thoracic cavity (pneumothorax, hydrothorax, empyema, &c.), and by obliteration of the bronchi.

§ 3. *Anomalies in Form and Position.*

Congenital anomalies in the *form* of the lungs are, for the most part, confined to some irregularity in regard to the lobes, which present a position of lateral inversion.

Amongst the acquired deviations, we must notice the displacement of the interlobular fissure in atrophia senilis, the pit-like depression of the surface in obsolescence of a portion of a lung, or in cases of cicatrization after loss of tissue, the flattening of a whole lung, or of a circumscribed portion, and the impression produced by the accumulation of gas in the thorax, by circumscribed exudations, aneurisms, or adventitious products.

Again, when the lung has been for a long time surrounded by a pleuritic effusion, and has acquired a thin, although resistant, fibro-serous investment which hinders its perfect re-expansion, it sometimes undergoes a singular change of form. It loses its sharp borders and the concavity of its base, and reminds us of the corresponding alteration of form presented by the liver after inflammation of its peritoneal coat. It sometimes, but not very frequently, happens that, if the pseudo-membranous investment be thinner at some spots than at others, the pulmonary parenchyma bulges out at those places into teat-like processes connected by a pedicle to the base.

Deviations in position, when *congenital*, are seen in the protrusion of the lungs through a wide fissure in the thorax, and in their lateral displacement. Under the *acquired deviations* we must notice the protrusion of the lungs in penetrating wounds of the chest, their displacement in various directions from dilatation of the abdomen and enlargement of its viscera, from copious effusion in the pericardium, from enlarged heart, from aneurism of the aorta, or adventitious structures in the mediastina, and generally from any kind of accumulation within the thorax. If there be no pre-existing adhesions in the last-named case to oppose the displacement, the lung, as has been already remarked, is constantly pressed inwards and upwards towards the mediastinum and vertebral column. A similar change in position occurs when obsolescence takes place from internal conditions, since the lung is then retracted on the bronchus.

§ 4. *Diseases of Texture.*

We shall commence with a description of two very simple alterations of tissue, which, although not very striking in themselves, lead to very important consequences, which, singularly enough, resemble each other; these are *rarefaction* (*vesicular emphysema*) and *condensation of the pulmonary tissue.*

a. Rarefaction of the Pulmonary Tissue.—Emphysema.—Under the term *pulmonary emphysema* we comprehend, according to Laennec, two different conditions, of which one (and by far the more important one) is not fairly entitled to this name; but this inaccuracy leads to no error, because, in using the terms *emphysema vesiculare* and *emphysema interlobulare*, we indicate the seats of the two diseases, and thus distinguish one from the other.

In *emphysema vesiculare* we have a morbid condition of the peripheral portion of the respiratory organs analogous to that which we have already described as dilatation of the bronchi, and even of the trachea. Had Laennec done nothing else for medical science, his discovery of this diseased condition, and of the causes giving rise to it, would have sufficed to render his name immortal.

Vesicular emphysema consists in a permanent dilatation of the pulmonary vesicles, and the respired atmospheric air is actually contained within them, and does not, as in ordinary emphysema, become extravasated into the interstitial texture.

It not unfrequently arises very rapidly as a vicarious development of pulmonary parenchyma in cases where a great portion of the lung has become impermeable; and it appears more especially to be produced in a high degree during the last moments of existence, as a consequence of the labored inspirations which then occur. Thus in diffused hepatisation we find the edges of the inflamed lobes puffy and emphysematous, and in the higher degrees of pulmonary tuberculosis, the same condition is observed in the interstitial parenchyma between the tubercles, and in the superficial stratum of the lung. In like manner it developes itself as a sequence of those acute and chronic diseases which prove fatal by paralyzing the nervous apparatus presiding over the chemical process, and in which there is the most laborious action of the respiratory muscles, deep inspiration, and an insatiable thirst for air; or it arises as a consequence of a sudden check to expiration, as in hemorrhages from the air-tubes, when the bronchi become obstructed by blood.

In such cases we observe the following appearances: the emphysematous portion of the lung is puffed up, and conveys to the hand a peculiar feeling, which may be compared to that of a cushion filled with air; is pale, varying in tint, from a palish red to a dull white color, and is perfectly anæmic; is dry, collapses rapidly on being cut, but on pressure crepitation is indistinct and dull; it floats on the surface when placed in water; its cells are more or less dilated, and their walls are attenuated in proportion to the rapidity with which the morbid change has been developed. Finally there is sometimes extensive laceration of the dilated cells, and the emphysematous portion of the lung then presents the appearance of a torn network swollen with air. This form of emphysema seldom attains this degree, except on the anterior edge and towards the base; and at these parts it gives rise to the escape of air beneath the pulmonary pleura, which consequently peels off from the lung.

This form of emphysema, in so far as relates to the attenuation of the walls of the dilated cells, presents an analogy with senile atrophy of the lungs.

Another form of vesicular emphysema developes itself slowly, gradually spreading itself over a large portion of the lung, till it finally involves the whole organ; it arises, in part, from other causes than those already mentioned, and constitutes a substantive disease of the lung, which, as Laennec remarks, unquestionably gives rise to most of the so-called nervous asthmas.

It presents many varieties in degree and extent. By degree, we refer to the extent of the dilatation of the pulmonary cells; it must, however, be remarked, that in emphysema of long standing, we always simultaneously find several degrees of dilatation, and that it is only during the commencement of the disease that the dilatation is observed to be uniform. The pulmonary cells may be dilated to the size of a millet-seed or pin's head, or to that of a hemp-seed, a pea, or even a bean, and, in proportion to the size which they attain, they deviate the more from their original shape. At first the disease is a genuine, simple dilatation of the cells, and when the cell-walls become to a certain extent thickened and rigid, it may be regarded as an active dilatation of the cells somewhat analogous to hypertrophy of the lungs. In higher degees, on the contrary, the dilatated cells unite to form larger spaces, their walls becoming atrophied by the pressure they exert on one another. Such hemp-seed, pea, or bean-sized cells always present a very irregularly sinuous, but on the whole a roundish form, and exhibit a singular arrangement; for on their inner surface there are elevated ridges, projecting to various heights within the cavities of the dilated cells, traversing them in various directions, and forming boundaries and imperfect partition-walls to the different sinuosities. We likewise perceive delicate threads, either extending across the cells, or hanging free in their cavities: these cover the elevated ridges and the remains of the contiguous walls of the pulmonary cells. The pressure exerted on the adjacent tissues, which gives rise to their atrophy, is proportional to the dilatation of the cells; and the cell-walls becoming thick and rigid, the emphysematous lung, when a section is made, either does not collapse at all, or collapses very slowly.

Moreover, this form of emphysema occurs most frequently, and is most highly developed, in the peripheral portion, and along the edges of the lungs: it is not unfrequently associated with bronchial dilatation; and this, amongst other signs, establishes the affinity of these two diseases. It either attacks a small portion of the lung only, being confined especially to the anterior edge of one or other of the upper lobes, or else it spreads over a whole lobe, or a whole lung, or even both lungs.

In cases of emphysema of both lungs, the association of all the anatomical signs presents the following picture of the disease:

Barrel-shaped dilatation of the thorax, with permanent depression of the intercostal spaces; great dorsal curvature of the spine; hypertrophy of the respiratory muscles; and a clear sound on percussion.

On opening the thorax, the lungs expand beyond the walls of the chest, are seen to be remarkably large, and do not collapse under the pressure of the atmosphere.

On their surface, and especially at their anterior edges, we find round prominences as large as a hemp-seed or a pea, either standing alone or arranged in groups, and which are nothing more than the dilated pulmonary cells which have been already described.

The lungs have a very peculiar, soft, elastic feeling, which may be compared to that of a cushion filled with down.

On being cut they collapse very slowly, and the air escapes sluggishly, with a very diffused sound, scarcely amounting to crepitation, and somewhat resembling that of air slowly escaping from a pair of bellows.

Their tissue is pale throughout, anæmic, and singularly dry.

When only one lung is emphysematous, then only the corresponding half of the thorax is dilated; but an important fact in this case is the displacement of the mediastinum and the heart towards the opposite side. Finally, if only individual portions of the lung are emphysematous, they may, if they are very numerous, and the disease is highly developed, prevent by their pressure the expansion of the neighboring healthy cells, and thus retain them in a state of persistent compression.

The conditions giving rise to the production of emphysema, and its pathogeny in general, although much labor has been devoted to the affection, are still far from clear. Laennec regards it as a consequence of his so-called dry catarrh with pearl-colored secretion, and explains it in a mechanical manner: this secretion, and the catarrhal puffiness of the mucous membrane, obstruct the bronchi in such a manner, that although they allow the inspired air to enter the pulmonary vesicles, the diminished energy of the act of expiration presents an impediment to its escape, and hence a portion of it is retained. In the succeeding inspirations new air is again conveyed to the pulmonary vesicles, whose dilatation is thus effectually accomplished, the expansion of the inspired air, from its elevated temperature, doubtless contributing to the result. Moreover, a prolonged retention of the breath during parturition, a stool, or in blowing wind-instruments, may give rise to emphysema.

In opposition to the view that emphysema is a consequence of catarrh, cases have been adduced in which either no catarrh had existed, or when it only followed the dyspnœa as a symptom of pre-existing emphysema; in reference to the last-named causes, it is alleged to have occurred in persons who have never been exposed to these diseases. Hence, a spontaneous dilatation of the pulmonary vesicles has been assumed, which at one time occurs as premature atrophy with attenuation of the cell-walls, while at another time, from equally unknown causes, it is associated, as in other hollow organs, with hypertrophy of the cell-walls.

Laennec's view regarding the former mode of development of the affection, is essentially an important one. We do not, however, believe that the long retention of the breath is, in itself, the principal cause of forcible expansion of the air-cells; we are much more inclined to attribute it to the very deep and forcible inspirations which at length follow the expirations, and, in illustration of this view, we may refer to the nature of the inspirations in croup, in the bronchial catarrh of children, and in hooping-cough. Besides inducing forcible dilatation, they may also cause a paralysis of the contractility of the pulmonary tissue, and, consequently, a stagnation of air in the dilated pulmonary vesicles.

Emphysema may, however, be developed in cases where none of these injurious influences have been present; and it may occur gradually in persons leading a sedentary life. In these cases the less frequent but the proportionally deeper inspirations are the more to be regarded, because they take place with very little action of the diaphragm (abdominal respiration), as the occupation of such persons requires a bent position compressing the abdominal cavity, and, at the same time, powerful exercise for the arms. A paralyzed and atrophied condition of the diaphragm is here of the greatest importance; for the hinderance to the abdominal respiration thus induced is compensated by the strained activity of the other great respiratory muscles; and this circumstance is in accordance with the facts that the dilatation of the thorax is most marked in its upper segment, and that emphysema is primarily and most fully developed in the upper lobes, and especially in their anterior portion.

The thickening of the walls of the dilated air-cells arises, in our opinion, principally from the tissue adjacent to them becoming broken down by compression, and fusing with the cell-walls themselves; nevertheless if the dilatations increase from the persistent pressure exercised by the dilated cells on one another, atrophy of the contiguous ~~walls will~~ ensue, and the cells will unite to form larger cavities, much in the same way as we occasionally observe to occur in ~~contiguous~~ bronchial sacs.

The dyspnœa, conditional on emphysema, depends on several causes.

α. The excessive accumulation of air in the pulmonary vesicles hinders the proper filling of the capillaries ramifying on their walls by the pressure it exerts on them, and thus interferes with the vitalization of a sufficient quantity of blood.

β. In the higher degrees of emphysema numerous capillaries become obliterated, not only in the walls of the dilated cells, but also in the surrounding atrophied tissue,—a condition which induces the above consequence in a still higher degree.

γ. The diminished contractility of the pulmonary tissue and the constantly labored inspirations which then become necessary in consequence of the imperfection in the chemical process of respiration, allow of only ~~a very imperfect emptying of the~~ pulmonary ~~cells and~~ consequently give rise to the permanent stagnation of ~~air no~~ longer fit for the purpose of respiration, which, in its turn, also tends to prevent a sufficient ventalization of the mass ~~of the~~ blood.

The impermeability of the capillaries depending on the two first-named conditions gradually, but unfailingly, leads to disease of the right side of the heart in the form of active dilatation, which proceeds to affect the venous system; the venosity and cyanosis which ensue from these changes constitute the leading grounds for the immunity of asthmatic persons from tuberculosis.

The impermeability of the capillary vascular system, moreover, gives rise to the anæmic state of emphysematous lungs, thus rendering it an impossibility that œdema, stasis, hemorrhage, or pneumonia, should be developed in them.

It is easy to understand how it proves fatal. It kills by finally inducing paralysis of the lungs, by asphyxia from the accumulation of air no longer fit for the process of respiration, by paralysis of the heart, or by vascular apoplexy of the brain.

Emphysema interlobulare is the only form which, strictly speaking, deserves the name of emphysema; it consists in an accumulation of air in the cellular interstices of the pulmonary lobules. It can only result from the rupture of one or more pulmonary vesicles, and the escape of air from them into the adjacent cellular interstices, if, indeed, we except the spontaneous development of gas into the interlobular cellular tissue, which is not altogether impossible.

We consequently find air-bladders in the cellular interstices, and especially on the surface; they vary in number and size, and are characterized by their paleness, transparency, and round or rather oblong form; they may be made to move in the direction of the interstices, and to run into one another, so as to form ridges which ramify in the same direction superficially, and into the body of the lung; sometimes they circumscribe and, as it were, insulate the lobules, and as they are broadest on the surface, and as their size diminishes in proportion to the depth to which they penetrate within the substance of the lung, they present a wedge-like shape. When they are very small and closely crowded together, they present the appearance of froth. On making a section of a portion of dried lung, we find the interstitial tissue presenting irregular cellular spaces of larger or small size, heaped, without order, on and around one another, and perfectly different from the adjacent air-cells. As has been already mentioned, most of the air is usually found accumulated in the peripheral interstices, so that the pleura presents a puffed up, vesicular appearance. The air often makes its way into the cellular tissue uniting the pleura to the lung, peeling off large patches, and forming flattish, convex, movable air-bladders; and, in these cases, it is to be feared that some of these bullæ may be ruptured, and that the air may be extravasated into the pleural sac. In other cases the extravasated air may penetrate into the substance and towards the root of the lungs, and pass into the cellular tissue of the mediastinum, and from thence into the neck, and thus cause general emphysema.

This condition usually co-exists with a puffy state of the lungs, but never with well-marked vesicular emphysema. It is most common in children; and is occasioned in them, as well as in the rarer instances in which it occurs in adults, by very rapid, deep inspirations, or by long retention of breath when great muscular exertions are made, requiring a fixed condition of the thorax. It is most commonly situated in the upper part of the lobes, and especially along their anterior edges.

b. Condensation of the Pulmonary Tissue.—A certain degree of condensation is natural to the lungs of children; it sometimes occurs in adults as an individual peculiarity, and is then often associated with smallness of the lungs and pleural sacs. It is also present as a transitory condition during pregnancy.

It only comes within the limits of pathology, when it has become permanent and highly developed, and offers a persistent impediment to the capillary circulation through the lungs.

Such a degree of condensation may arise when the abdomen becomes enlarged and encroaches on the thoracic cavity, but in children it is more frequently dependent on lateral depression of the thorax consequent on atrophy of the great respiratory muscles, or on rachitis affecting the chest; it may also arise from spinal curvature, distension of the pericardium, enlargement of the heart, large aneurisms, adventitious products, &c.; or from the pressure exerted by an accumulation of air or fluid in the cavity of the chest, from pleuritic exudation, or from bronchial dilatation; and, according to the various exciting causes, it may occur simultaneously in both lungs, or only in one, or merely in certain portions of the pulmonary tissue, as in cases of rapidly developed emphysema, where we not unfrequently find single lobules compressed in the centre of the emphysematous portion, or in cases of atrophy of the external respiratory muscles, where single circumscribed portions of lung are found in a state of condensation under the bent anterior ends of the ribs.

There are different grades varying from simple increase of density characterized by augmented consistence and compression of the pulmonary tissue, and by a stasis and hyperæmia depending on obstructed circulation, to such a degreee of compression as to destroy the air-cells, to arrest the capillary circulation, and to give rise to atrophy of the texture of the lung.

The most intense compression of the lung occurs in cases where there is abudant pleuritic effusion. With the alterations in position and form, to which allusion has already been made, the lung always becomes denser and gradually becomes impermeable to air, and, finally, even to the passage of blood along its capillaries. If the lung still contains blood, its red color gives it such a similarity to flesh, that this condition has received the name of *carnificatio pulmonis*, but at a subsequent period it becomes of a dirty brown, or, more commonly, of a bluish-gray or lead-color, and is tough and leathery, and sinks in water.

If the state of extreme compression persist for a length of time, the pulmonary tissue finally becomes obsolete, that is to say, it becomes converted into a cellulo-fibrous tissue,—a condition altogether distinct from atrophy of the pulmonary tissue.

Excessive condensation of the lungs gives rise to consequences similar to those of emphysema; it impedes the capillary circulation, and thus occasions stasis in the trunk of the pulmonary artery, giving rise to active dilatation in the heart, and consequently to venosity and cyanosis. Hence, like emphysema, it affords a remarkable immunity from tuberculosis, especially when associated with curvature of the spine.

There is a peculiar form of anomalous condensation of the pulmonary tissue, probably dependent on a congenital bronchial catarrh or catarrhal pneumonia, and consisting in a deficient development of the lungs of new-born children, in which certain portions of those organs retain their fœtal condition after birth. It is termed *atelectasis* of the lungs, and presents various degrees of obstruction to the closure of the fœtal passages, namely, the ductus arteriosus and the foramen ovale, thus giving rise to predominance of the right side of the heart and to cyanosis.

c. Hyperæmia; Stasis—Apoplexy of the Lungs.—No organ with the exception of the brain, is so frequently the seat of hyperæmia as the lung. It occurs in various degrees, and developes itself either gradually or with intense rapidity, and is the anatomical basis of most sudden deaths.

In a lesser degree, as simple hyperæmia, it is frequently an habitual, and not rarely a periodic affection of an active nature; it often ensues with great rapidity, and may prove speedily fatal by itself, or more frequently by the superaddition of acute œdema. We then find both lungs uniformly puffy, and of a dark-red color; their vessels, even to the capillaries, being filled with dark blood, and their tissue being succulent and softened, but still crepitating. In the bronchi we find a grayish, sometimes reddish mucus mixed with air-bubbles. The heart is usually somewhat dilated, and always contains a large quantity of thin liquid or slighty coagulated dark blood, especially in its right cavities. The veins of the membranes of the brain are usually full to distension, and serous effusion into the cerebral ventricles frequently occurs as a consecutive complication. The outer surface of the body is characterized by livor, and by the rapid occurrence of extensive and very dark-colored death-spots; the face in particular is very puffy, and of a more or less bluish tint; the eyes and mouth are generally more or less open, and the conjunctivæ injected; the mucous membrane of the mouth is livid, and that of the throat is covered with tough mucus. Grayish or pale reddish, frothy mucus is found in the trachea.

In a higher degree hyperæmia amounts to *stasis*. In this stage the parenchyma of the lung is of a purple or black-red tint, and, as it were, saturated with blood; and as it somewhat resembles the substance of the spleen, this condition has received the name of *splenization*. Several of the other characters of this condition are subject to various modifications, depending primarily on the degree and the duration of the hyperæmia, but to a lesser extent on the nature of the stasis and the composition of the blood. When it is recent and comparatively slight, the parenchyma is denser, but easily torn; it crepitates, although less clearly than in the normal state; on cutting it, a large quantity of fluid blood escapes; the diseased portion is puffy, and floats on water. In a higher degree, and when the stasis has continued for a longer period, the walls of the air-cells and the interstitial tissue become swollen, so that the former may become perfectly impermeable to air: the parenchyma consequently become denser, hard, and heavy, and ceases to crepitate; and on making an incision only a comparatively trifling quantity of thick fluid blood escapes. The blood appears, as it were, fused into the tissue of the lung, the whole affected portion having a somewhat shrunken appearance.

The blood contained in the splenified portion of the lung presents various shades of discoloration, viscidity, fluidity, or gaseity, according to the nature of its composition and the character of the stasis.

According to circumstances we occasionally find in the bronchial tubes either a sanguineo-mucous or sanguineo-serous fluid.

Stasis is the result of an active or passive, or of a mechanical hyperæmia; either of these may prove fatal by itself, especially when extensively developed; and either may sooner or later pass into inflammatory stasis and inflammation. It never attacks both lungs simultaneously and in an equal degree; it generally exhibits a preference for the lower lobes, and when it extends over a whole lung, usually commences inferiorly.

Stases of a *passive* nature in the most dependent posterior and inferior portions of the lungs, such as are developed in bedridden old persons, or in individuals confined to bed for a length of time in consequence of cerebral disease, typhus and typhoid affections, any adynamic diseases, and especially paralyzed conditions of the lungs, are important. They constitute the *pulmonary hypostasis* of Piorry.

Mechanical stasis is most commonly dependent on organic diseases of the heart, although excessive density of the lungs may give rise to it.

It is developed, according to circumstances, either in the arterial or the venous portion of the capillary system of the lungs.

It is of great importance to distinguish, as clearly as possible, between these conditions and the stasis which is developed in the body after death,—cadaveric hyperæmia of the lungs, more especially as this is very frequent and is often combined with the former. The latter is always most marked at the posterior portion of the lungs, gradually diminishing in the superior and anterior directions. The lung is soft and crepitates, and apparently is not so much saturated with actual blood, as with a sanguineous, dark-red, frothy, discolored serosity, which is poured forth in abundance from the cut surface, and may be entirely removed by moderate pressure, after which there remains a pale, discolored parenchyma compressed in proportion to the pressure employed. In consequence of prolonged imbibition, the pleural sacs not only become discolored, but a certain quantity of sanguineous discolored serosity makes its way into their cavities.

Different views have been held, especially in recent times, since doubts have been entertained regarding Laennec's theory of hæmoptoic infarctus, respecting the relation which these conditions bear to pulmonary hemorrhage and apoplexy of the lungs. For our own part, on the one hand, we regard them as representing *hyperæmia* of a lower or higher degree, which, under certain conditions, may give rise to hæmoptysis, and, on the other hand, it appears clear to us that they also represent *apoplexies*, namely, in a less degree a vascular apoplexy, and in a higher degree an apoplexy with effusion of blood into the parenchyma of the lungs; but as the blood is not at all events originally effused into the cavities of the air-cells, apoplexies are *not necessarily* associated with hæmoptysis, and hence must be distinguished from it.

The latter variety, namely, hemorrhage into the cavities of the air-cells, corresponds with Laennec's *pulmonary apoplexy or hæmoptoic infarctus*. Our own experience confirms the views of that great physician regarding the existence of this morbid change, and the manner in which it is produced. When highly developed, it is attended with laceration of the texture of the lungs.

The apoplexy of Laennec is characterized by the following signs:—

We find blackish-red patches in the substance of the lungs, which attract attention not only by their color and consistence, but also by their definite outline. On examining the cut, or, what is better, the torn surface of the diseased portion, we observe it to be more or less coarsely granular and dry, the granulation being often very irregularly distributed. The tissue itself is tough and yet easily torn, and presents throughout, both at the centre and at the periphery, the same consistence. The whole represents an effusion of blood into the cavities of the air-cells, which distends them to a certain extent and then coagulates within them, thus giving rise to the granular texture of the hæmoptoic infarctus. The interstitial tissues are compressed, and infiltrated with blood, and hence the color of the diseased part is uniform throughout. The terminations of the bronchial tubes are also filled with the extravasated blood, their walls being reddened by imbibition, just as we observe in the case of bloodvessels. On scraping the infarctus with the back of a scalpel, there is poured forth a very slight quantity of thick blood intermixed with numerous, black, grumous flocculi.

Hæmoptoic infarctus bears the greatest similarity to red hepatization of the pulmonary tissue; none but very inexperienced persons can, however, mistake one for the other, for each of the above properties of infarctus presents a distinguishing sign from hepatization. These, briefly summed up, are the well-defined limitation of the infarctus, the homogeneity of its consistence and color throughout its whole extent, the *coarse and irregularly granular* appearance and the dry fragility of its cut or torn surface, and the nature of the product obtained on pressing or cutting its surface.

The pulmonary tissue in contact with the infarctus is either in a perfectly healthy condition or else in a state of some other pre-existing or consecutive disease; in every case it is clearly separated from the infarctus. Amongst the pre-existing diseases we must especially mention tuberculosis and pneumonia, whilst the most common consecutive affections are emphysema and œdema of the lungs.

It occasionally happens that this limitation of the hæmoptoic infarctus cannot be detected without a somewhat close examination. This is the case when the parenchyma surrounding it to a certain distance, is the seat of an effusion of fluid blood, whose limitation is by no means sharply defined, since it changes towards its periphery into a palish, sanguineo-serous infiltration, and thus gradually loses itself in the normal tissue. Still, by a careful investigation, we may discover the infarctus seated within the fluid effusion, and plainly separated from it by its consistence and darker color.

The size of the hæmoptoic engorgement is seldom very great, for while, as Laennec observes, it scarcely ever exceeds four cubic inches, it is frequently less than one. We often find only one infarctus; sometimes, however, several are simultaneously present in one or both lungs.

They are deeply seated in the parenchyma of the lungs, near their roots, or in the posterior portion of the lower lobes; they are, however, occasionally found near the surface, and may be recognized through the pleura by external inspection. It sometimes happens that when they have existed for a considerable period, the pleura above them becomes inflamed.

They are often, but by no means always, accompanied by considerable hæmoptysis; their size stands in no relation to its amount, indeed there may have been very considerable hæmoptysis, without a trace of hæmoptoic infarctus being perceptible after death; thus, when the effused blood has coagulated rapidly and completely in the pulmonary cells, notwithstanding the hæmoptoic infarctus, there will be no hæmoptysis; in another case the blood does not coagulate at all, but is coughed up in a fluid state, and then, notwithstanding the hæmoptysis, there is no hæmoptoic infarctus; or, again, the primary effusion may coagulate and form an infarctus, while hemorrhage in the surrounding parenchyma may be the source of hæmoptysis (see above).

This form of apoplexy is very frequently found to be associated with active dilatation of the right side of the heart, and it seems to bear the same pathogenetic relation to this cardiac affection as cerebral apoplexy bears to active dilatation of the left side of the heart.

In recent times some doubts have been suggested regarding the true connection between hæmoptoic infarctus and pulmonary hemorrhage, and it has been regarded as the result of hemorrhage of the finer bronchial ramifications, that is to say, as depending on the coagulation of the blood which has escaped from the bronchi into the pulmonary vesicles. Although we fully believe that this may sometimes be the case, yet in the absence of any positive proof we prefer adopting Laennec's view, in relation to the assumed bronchial hemorrhage, for the following reasons: (1), because hæmoptoic infarctus very often occurs without hæmoptysis, while a bronchial hemorrhage could hardly take place without any sanguineous expectoration; and (2), because if, as is reasonable, we recognize the influence of hypertrophy of the right side of the heart, we shall see that this influence, notwithstanding the anastomoses of the two systems of vessels, is especially exerted on the pulmonary arteries, and that it will thus serve to elucidate true pulmonary hemorrhage.

When this form of apoplexy is very much developed it is accompanied with *laceration of the pulmonary tissue;* we find a cavity in the lung similar to those which are met with in cerebral apoplexy, and containing a certain quantity of more or less coagulated blood. The surrounding pulmonary texture is torn, suffused with blood, and presents, to a certain degree of thickness, an appearance of hæmoptoic infarctus.

The position of these cavities coincides with that of the hæmoptoic infarctus; it has, in rare cases, happened that when situated in the peripheral portion of the lungs, they have opened by a rent into the pleural sac, thus giving rise to the free effusion of blood into that cavity, and to pneumothorax.

The size of these cavities varies, but it scarcely ever exceeds that of the hæmoptoic infarctus. Gangrene of the lungs sometimes, however, gives rise to very considerable accumulation of blood.

Simple hyperæmia and stasis are easily reduced to the normal state, especially under proper and judicious treatment; but they leave a great predisposition to relapses, and hence they usually require a prolonged prophylaxis.

The following questions suggest themselves:—what alterations do hæmoptoic infarctus and apoplexy with laceration undergo in the progress of time? and in what way is the tendency to cure and its successful accomplishment evinced?

It is only very rarely that experience presents us with pure indisputable facts bearing on the various stages of the healing process, necessary for the solution of these questions; still from the scanty materials in our possession, and by a comparison with analogous processes in other organs, we arrive at the following conclusions:

The effusion in hæmoptoic infarctus either (1) quickly becomes fluid, assumes a blackish-brown, rusty, and wine-lees tint, and in this state is partly absorbed and partly excreted through the bronchi (thus, doubtless, causing the peculiar expectoration sometimes observed to follow hæmoptysis), the parenchyma remaining for a time moist, soft, lacerable, and of a rusty or wine-lees color, and gradually returning to its normal ate; or (2) the effusion is only partly removed in this manner, and ere remains a tough fibrinous coagulum, which gradually becomes per-ctly decolorized, or a loose glutinous coagulum, saturated with black igment, the surrounding parenchyma becoming shrivelled up, and de-enerating into a cellulo-fibrous tissue of either a white or a blackish nt.

Apoplexy with laceration heals, after the absorption of the effusion, ither by a direct agglutination of the walls of the sac, or by the con-action of the parenchyma round a fibrinous coagulum, which finally ecomes cretified, or by the conversion of the parenchyma into a cellulo-brous capsule, enclosing a glutinous coagulum, consisting for the most art of pigment.

d. Anœmia of the Lungs.—There are various conditions which may ive rise to a deficiency of blood in the lungs. It may depend:

a. On exhausting hemorrhages.

b. On wasting of the blood, consequent on various acute and chronic iseases.

c. On the inspissation of the blood, consequent on rapid and great ss of serum, and on the inability of the blood, in this condition, to nter the capillaries; this is especially the cause of the anæmia of the ngs in Asiatic cholera.

d. Finally it occurs in association with pulmonary atrophy, with em-hysema, and with the higher degrees of compression of the lungs.

e. Œdema of the Lungs.—Pulmonary œdema is a very frequent and xtremely important disease. Its essential and primary symptom is the filtration of the parenchyma with a serous fluid, which is obvious even om an external inspection, and much more so on examining the inte-ior of the viscus, which pours forth a serous fluid when a section is ade into it. The serum, however, does not vary only in regard to its uantity (that is to say, not only are there differences in the degrees of e œdema), but it likewise presents many differences in relation to its roperties.

In order to understand the importance of pulmonary œdema under all onditions, it is necessary for us to direct attention to the information hich we have acquired from clinical observation, and from careful ex-mination and experimental investigation of the dead body in relation the seat of the serous effusion. We thus ascertain that the serum is ffused into the cavities of the air-cells, where it accumulates, either lone or mixed with varying quantities of air, according to circumstances. rom hence it flows in greater or less quantity, either mixed with air nd frothy (as bronchial foam), or unmixed with air, into the bronchial bes. The walls of the air-cells and the interstitial tissue are also ore or less saturated and infiltrated with serum, but the true seat of e fluid which so often escapes in astonishing quantities from the cut urface of the parenchyma of an œdematous lung is in the air-cells and he bronchial canals.

Pulmonary œdema occurs both in an *acute* and in a *chronic* form, and etween these extremes there are many transition stages presenting mere shades of difference. In *acute œdema* the lung appears swollen, does not collapse, feels puffy, and when we press it with the finger we detect a fluid which escapes with a crackling noise; its elasticity is only slightly diminished, so that scarcely any perceptible pitting remains after the pressure; it is of a pale reddish colour, very pale and deficient in blood when anæmia is present, and more or less red and congested if there be hyperæmia; the serum which is effused from the cut surface is mixed with much air, which renders it frothy, and is usually of a pale red color; but in œdema arising from prolonged stasis and simultaneous decomposition of the blood, it is red and discolored, having an icteric tint. The parenchyma is softer than usual, very moist, singularly yielding, and easily torn.

If the œdema lasts *for a longer time*, the pulmonary tissue gradually loses its elasticity, the lung pits more distinctly on pressure, becomes paler, assumes a faded, dirty gray color, and becomes opaque and dull; the air is gradually pressed out of it; it crepitates less, when cut; and the serum is less frothy, gradually loses its color, and becomes clear and limpid. The parenchyma becomes gradually infiltrated with serum, the walls of the air-cells and the interstitial tissue become swollen, and hence the lung becomes denser and more resistant.

Finally, in cases where *chronic œdema* has been very fully developed from its commencement, the lung appears pale, of a dirty gray color, anæmic, not swollen, but heavy, dense, and resistant, pitting on pressure and no longer crepitating; a grayish or somewhat greenish serum unmixed with air flows from the cut surface. Dropsical accumulation in the pleural sac is almost always simultaneously present.

Œdema of the lungs, like acute œdema of the glottis, is often *very rapidly* developed; from an active hyperæmia or a passive or mechanical stasis, it quickly reaches a high degree of intensity, extends simultaneously over both lungs, and in a short time causes death by suffocation. This is frequently the cause of the suffocation of adults and of new-born children, and is often combined with hyperæmia and serous effusion within the cavity of the cranium. The dead body usually presents the same appearances as those which we have described as occurring in pulmonary apoplexy; the lungs in particular exhibit œdema, and a frothy serous fluid is accumulated in the bronchial passages, which is frequently seen as a thick, white, or whitish-red froth, at the oral and nasal cavities. It may also be developed as a consequence of acute or chronic bronchial catarrh, or of exudative processes (croup) on the tracheal and bronchial mucous membranes; it is a constant symptom in acute pulmonary tuberculosis, in acute decompositions of the blood and after the retrogression of erysipelas, scarlatina, variola, rheumatism, miliaria, &c. In the form of more or less developed acute œdema it accompanies the various stages of pneumonia and the metastases: and is associated with hæmoptoic infarctus, with pulmonary cancer, and especially with pulmonary tuberculosis. Lastly, it appears as a consequence of cerebral diseases, of general anæmia and tabes, and occurs towards the end of almost all chronic diseases.

Chronic œdema, moreover, exists with general dropsy, with dropsy of the great serous sacs, with chronic diseases of the heart and great vessels, &c. It is rarely an idiopathic and independent disease.

The extent of œdema is various; the very acute and rapidly fatal œdema generally attacks both lungs almost equally; in other cases it is limited to individual portions of them. The œdema in cases of pneumonia commonly affects the circumference of the inflamed part; that which occurs as a consequence of chronic diseases, for the most part, attacks the posterior and inferior parts of the lungs, which are most exposed to the influence of gravitation.

f. Inflammations of the Lungs (*Pneumoniæ*).—Pathologists are in the habit of recognizing only one form of pneumonia. It is true that this is by far the most frequent form; but even in regard to this there are several points in which we cannot agree with the accepted view. We may provisionally and very briefly remark that the evidence of its croupous nature will be the more manifest in proportion to the epidemic constitution and the special cause of the disease, the rapidity of its course, the degree of its intensity, &c. We shall treat of this, the most common form of pneumonia, under the designation of:

1. *Croupous Pneumonia.*—The course of this disease is divided, as is well known, into three stages, which have received the names of *inflammatory engorgement*, *hepatization*, and *purulent infiltration.* We shall first consider the case in which a whole lung, or at least a whole lobe is affected.

The first stage, *inflammatory engorgement*, is always preceded by the above described condition of simple stasis and splenization of the parenchyma; but, conversely, this condition is not always developed into inflammatory stasis or engorgement. This affords the explanation of the contested question regarding the inflammatory nature of simple stasis and its significance as a stage of the inflammatory process. It is only by a careful examination that we can distinguish inflammatory engorgement from simple stasis. The lung is generally of a dark red color, heavy and tough; it pits on pressure, and we perceive that it contains a fluid and little or no air. On cutting it we find its substance denser than in the normal state, in consequence of the swollen condition of its tissues and of its being filled with a sero-sanguineous fluid; and according to the degree of this state the lung may either crepitate and swim in water, in consequence of its still containing a little air, or it may sink and not crepitate; it is easily torn, very moist, and pours forth a sero-sanguineous fluid, which is sometimes rather frothy and sometimes not at all so.

This condition has, as we have already remarked, the greatest similarity and affinity to simple stasis, especially when the latter is combined with œdema.

We will now direct attention to the characteristic symptoms by which inflammatory engorgement may be distinguished from the above-named similar condition. Amongst them we may mention the *color tending to a brownish-red*, and the *moisture* of the parenchyma, which in itself is sufficient to distinguish the inflammatory from the simple stasis, and also from that combined with œdema, by the special circumstance of its depending on the tissue being filled with blood that has already undergone the inflammatory metamorphosis, or, in other words, with a brownish or brick-red, thin but viscid fluid mixed with black, crumbling flocculi. As soon as the transition to the second stage commences, there is a *secretion* of a very viscid, tough, reddish-brown fluid,—the characteristic sputum, as may be proved by an examination after death; and, finally, there is the true exudation with which appears—

The second stage, or that of *hepatization*, in which the lung appears both externally and internally of a dark brownish-red color, is solid but friable, does not crepitate, and sinks when placed in water. On examining the cut surface we either observe the above color uniformly distributed, or it is deposited in the form of irregular spots amongst the black pulmonary tissue; while the pale red interlobular tissue presents ramifications, and the whitish bronchial tubes and the bloodvessels form stripes or islands which destroy the uniformity of the coloring, and give the cut section a marbled appearance. Further, the cut or torn surface presents a change of texture which is perfectly characteristic; when the light falls obliquely on this surface we perceive that it has a granular appearance, which is the special reason why it resembles the tissue of the liver, although the similarity is aided by the firmness, fragility and color of a hepatized lung. Hence the origin of the term *hepatization*, which is now generally adopted and understood. The character of the granulation is uniform, and the individual granules are roundish.—Scarcely anything exudes from the cut surface, and it is only by a certain amount of pressure, or by passing the scalpel over it, that a brownish-red, turbid, sanguineo-serous fluid escapes, mixed with blackish-brown and a few reddish-gray flocculi.

The volume of the hepatized lung does not in general exceed that of the healthy lung in a state of full inspiration; hence its surface is smooth, and never indented by the ribs, and there is no dilatation of the thorax. Sometimes, however, we find single lobules projecting higher above the surface than others, in consequence of a want of uniformity in the progress and the degree of the exudation; and the granulation of these tissues is coarser in consequence of the products of inflammation being here deposited in greater quantity than in other parts.

This form of hepatization is named the *red*, to distinguish it from those varieties in which this color is no longer present, although the granular texture remains.

On what does the granular texture of the hepatized lung depend? This is a most important question; the ordinary answer to which is, that it results from so great a swelling of the walls of the air-cells that their cavities become obliterated, each granulation being thus represented by an air-cell. We can by no means give our assent to this generally received opinion, for we are convinced that the granulations are produced by the inflammatory product deposited in the cavities of the air-cells; we shall, however, postpone bringing forward our evidence on this point, since the perfect solution of the question is intimately connected with the determination of the seat and nature of the pneumonic process.

Each granulation is a hardish, fragile, dark-red, roundish plug, which adheres so closely to the dark-red, swollen wall of the air-cell, that it is 'difficult to separate and extract it.

Pneumonia passes from the stage of red hepatization through several scarcely distinct transition-stages till it finally attains the true *third* *stage.* These transition-stages are characterized by alterations of consistence and especially of color. The red, hepatized lung gradually becomes paler, assumes a brownish-red, then a grayish-red or gray, and finally a yellowish color, and thus presents the condition to which the term *gray hepatization* has been appropriately given. We can recognize this coloration externally, but far better on examining a cut surface; and we can perceive that in many cases, the tint is not monotonous, but that the black pulmonary tissue is more or less uniformly sprinkled over the grayish-red, gray, or yellowish-gray ground, which is also marked by the white projecting cut vessels, so that the whole presents a granite-like appearance.

The granular texture is still present, and even becomes decidedly more distinct at the commencement of the third stage, especially when the progress of the disease has been rapid and tumultuous; the consistence diminishes and the decoloration increases the nearer the disease approaches to the third stage; although the lung feels tolerably firm, it remains pitted after pressure, and is yielding and easily torn, and a grayish-red, very turbid, flocculent, viscid fluid exudes from its cut or torn surface.

If we examine the granulations in these transition-stages, we perceive that they have become more marked, larger, and more independent of the surrounding structures; and that they can be more easily separated and removed, as they only adhere loosely by a glutinous substance to the walls of the cells.

Third Stage, Purulent Infiltration.—At its commencement the change of color of the hepatized tissue to a yellow tint (to which we have already adverted) becomes more or less uniform, the granular texture very rapidly disappears, and is succeeded by a purulent infiltration of the parenchyma. The lung then becomes heavy; any pressure on it forms and leaves a distinct pit; the cut surface is yellow or straw-colored, with interspersed spots of black pulmonary tissue, and effuses a large quantity of a very viscid, purulent fluid of the same color as the surface, and of a sickly odor; the parenchyma is extremely yielding, gives way on the slightest pressure, so that if not carefully handled, cavities are easily formed in it, which are the more likely to be taken for abscesses, as they actually are very similar to fresh accumulations of pus. The granular texture has now altogether disappeared, and, on removing the pus from a piece of lung by careful pressure and washing, we perceive that its substance has again assumed its spongy, cellular tissue.

The bronchi present several changes, especially in their final ramifications; in the first stage their mucous membrane is reddened and swollen, subsequently however it becomes paler; and they almost always contain first a reddish, and afterwards a whitish, purulent, fluid exudation. The vessels are frequently clogged by exudations of this nature.

These are the three stages through which well-marked cases of acute pneumonia run; the last is the ordinary and natural mode of termination, and is frequently although by no means necessarily fatal, for, partly by expectoration and partly by resorption of the pus, the lung may return to its normal condition. There is no other and earlier stage than that which we have described as the stage of stasis, for the condition described as such by Stokes is in no respect inflammatory. The bright-red color of the lungs or of portions of them, which Stokes regards as the earliest stage of inflammation, and attributes to arterial injection, is;—

(*a*.) Always dependent on anæmia, which is frequently very highly developed.

(*b*.) The lungs, or the affected parts of them, are puffed up, but are devoid of turgor and resistance in consequence of their capillary vessels not being duly filled; they collapse readily, and not a trace of a swelling of the tissue remains.

(*c*.) This condition always occurs when, in consequence of paralysis of the heart or of excessive thickness of the blood, the capillaries of the lungs can no longer be injected, and the little blood occurring in them is repeatedly exposed to the chemical influence of the atmospheric oxygen by the inspirations during the death-struggle. In this way we observe this condition either distributed over large portions of the lungs, or confined to small spots of lungs otherwise healthy, or associated with hyperæmia and stasis in many cases of asphyxia in new-born children and adults, in consequence of rapidly exhausting diarrhœas, of Asiatic cholera, after extensive burns of the general integument, &c.

Before entering into any further discussions, it will be most expedient that we should add to the above sketch the conclusions regarding the seat and nature of the pneumonic process, at which we arrive from an accurate anatomical investigation after death, and a review of the physical phenomena during life. These conclusions will not only find an influential application in what is to follow, but will also receive corroboration from it.

In relation to the first point, we have already stated, in our remarks on the formation of the granular texture of the hepatized lung, that the granulations are formed by the inflammatory product deposited in the cavity of the air-cells. Their formation, or, in other words, the exudation, is preceded by the secretion of a viscid, tough, reddish-brown fluid in the cavities of the cells, which gives rise to the crepitation well known to auscultators: as the stage of hepatization advances this fluid disappears, and the air-cells become filled with plastic exudation. The granulations are roundish, and at first of a dark-red color, hardish, and fragile; they appear to have uniformly coalesced with the swollen, dark-red walls of the cells, from which it is difficult to isolate and extract them. The inflammatory turgor and the redness of the tissue become then moderated; the granulations become paler, of a grayish-red, and finally a grayish-yellow tint, while they appear less dense in structure, and become somewhat swollen. The secretion of a glutinous mucus is established around their circumference, which loosens their connection with the cell-wall, thus rendering themselves and the swelling more obvious: they appear surrounded by a light reddish cell-wall, and their distinctness is proportional to its paleness. Finally, they break down into a purulent fluid, mixed with this glutinous, mucous secretion. Hence the *seat* of the pneumonic process is on the walls of the air-cells—that is to say, on the *pulmonary mucous membrane*, and its product is deposited in the cavities of the air-cells; from this period—that is to say, from the stage of red hepatization—the process consists in a metamorphosis tending to the fusion and breaking down of the exudation, under the influence of an inflammatory process, which is now declining in intensity. These conclusions are further strengthened by the following considerations:

(*a.*) If the granulations were regarded as swollen, and consequently obliterated, air-cells, they could neither exhibit the above anatomical relations, nor could they present the metamorphoses which, regarding them as inflammatory products, we have represented that they undergo; that is to say, if we take an unbiassed view of the subject.

(*b.*) Even the greatest swelling of the air-cells could not modify the volume of the hepatized lung, while our theory perfectly explains this phenomenon.

(*c.*) If the third stage, or that of purulent infiltration, were a suppuration of the interstitial tissue, a recovery from it without abscess and solution of continuity could not be possible; whereas it takes place by partial expectoration and partial absorption of the dissolved exudation, without any ulcerous destruction of tissue, in which case anatomical investigation shows, that, in purulent infiltration of the lungs the texture is altogether undestroyed, and of a spongy, cellular nature.

(*d.*) Finally, the same process, as a general rule, extends to the terminal ramifications of the inflamed lung.

Even from what has been already said, it appears that, in relation to its anatomical elements, we may regard pneumonia as *a croupous process on the pulmonary mucous membrane*, or, in other words, as *a parenchymatous croup*. It exhibits, even within the limited circle of its anatomical relations, a perfect identity with the croupous process on other mucous membranes; we shall, however, subsequently develope this view in a more extended and general manner.

We very often find the three stages coexisting, and can observe all the transition-stages passing into one another. Purulent infiltration and gray hepatization generally predominate in the central and inferior part of the inflamed lobe; there is grayish-red or red hepatization towards the periphery; above this there is inflammatory engorgement; while, finally, simple stasis, and very frequently acute œdema in different stages, are present in the adjacent tissue.

Pneumonia may prove fatal in any of these stages; it may also retrograde from each to the normal condition. Besides the above-described termination in purulent fusion of the inflammatory product (purulent infiltration), it may in rare cases give rise to abscesses or induration, or may end in other ways, which can be more suitably noticed in a future part of the work.

If the pneumonia has reached the third stage, and is proceeding towards a cure, we observe the following phenomena:—The purulent fluid is gradually removed, and an exhalation of serum commences from the pulmonary mucous membrane; the pus which still remains is gradually rendered thinner by this admixture, and is finally converted into a flocculent, turbid serosity, which becomes mixed with air-bubbles as soon as the air again begins to penetrate. The parenchyma, at the same time, becomes paler, and of a grayish-yellow tint, and retains this color for a considerable time; it crepitates less distinctly than in the normal state, is softer and moister, is more or less œdematous, and easily torn.

The lung can also retrograde from the second stage, that of hepatization, to the normal state, without the purulent liquescence of the exudation. This process is undoubtedly one of the most difficult which the healing powers of nature can accomplish; for it always takes place somewhat slowly, and undoubtedly the more so in proportion, on the one hand, to the plasticity of the product, and, on the other, to the exhaustion following the effusion, whether the exhaustion be dependent on the disease, or be induced by the activity of the treatment. The granulations, together with the tissue, gradually become paler, and a serous fluid, which is secreted in the cells, seems by degrees to cause a fusion of the granulations, layer by layer. The tissue still retains a granular character, but the granulations always become smaller, of a pale red or reddish-gray color, and are bathed in a serous fluid, which is mixed with tolerably consistent, pale reddish or whitish flocculi, and which gradually becomes frothy from the entrance of air. When the granulations are thus finally melted down, the parenchyma remains for some time in a state of serous infiltration, and is redder, firmer, and more resistant than in the normal state, owing, apparently, to a still existing infarctus of the walls of the air-cells and of the interstitial tissue. This retrograde process does not go on with equal or uniform rapidity at all parts; and we can often confirm our diagnosis by finding dense and still hepatized patches in tissue which has more or less returned to the normal state.

Finally, pneumonia retrogrades from the first stage—that of inflammatory stasis—to the normal condition; this is very frequently the case when those favorable influences are present which it is the great object of the healing art to induce. The inflammatory stasis, after it has deposited a moderate infiltration of turbid serous fluid, is converted into simple stasis, and after this is resolved, the tissue again becomes normal, but remains for some time the seat of hyperæmia which may easily relapse into inflammatory stasis.

Pulmonary Abscess.—We have already described the termination of pneumonia in purulent infiltration, that is to say, in purulent solution of the inflammatory product, which occurs without any separation of continuity or ulcerous destruction. The reverse takes place when accumulations of pus are formed in the lung. This termination of pneumonia is extremely rare; but this rarity need not excite our wonder, nor do we require the explanation attempted by Laennec, if we adhere to our view of the pneumonic process. The conditions giving rise to the formation of pulmonary abscesses and the mode in which it is formed are, however, little known. Of all the theories which have been advanced, that is most conformable with the nature of the pneumonic process, which regards it as a consequence of a peculiar character of the inflammatory process, causing the pulmonary mucous membrane, which has been deprived of its epithelial investment, and the other tissues entering into the composition of the parenchyma, to become disintegrated and to suppurate,—a process analogous to that which occurs in many cases of true croup of the mucous membrane, and still more so to other exudative processes occurring on the same structure.

A recently formed, fresh pulmonary abscess presents the appearance of a cavern of irregular form filled with pus formed from the disintegration of the lung and surrounded by a softened parenchyma infiltrated with pus, and in some places hanging in shreds. It is perfectly similar to those rents which may be produced by pressure, when we are carelessly handling a lung in the stage of purulent infiltration, or on attempting to separate it from adhesions to the costal wall, and which we have already warned our readers against mistaking for pulmonary abscess.

The abscess either enlarges in the same way in which it originated, by the continued solution of the inflammatory product and of the tissue of its walls, or else by the confluence of other neighboring abscesses. As a general rule the suppuration extends over the whole of the inflamed portion of the lung, and hence the abscesses consequent on the lobular inflammation (of which we have already spoken) are always very considerable. According to their size we observe one or more bronchial tubes opening into them with transverse or oblique mouths, and their tissues also become the seat of purulent solution. These abscesses represent the true but very rare *ulcerous pulmonary phthisis* which is based on inflammation.

It proves fatal either by the supervention of fresh pneumonia around it, or pleuritis, or by the absorption of pus into the blood, with the symptoms of pyæmia and hectic fever. In rare cases it perforates the pulmonary pleura, and causes suppuration of the adjacent tissues, after having given rise to pleuritis and adhesion of the lung to the wall of the chest. Finally, in some very rare cases, it opens freely into the thorax before pleuritis and adhesion of the lung to the walls of the chest have been established; a general or circumscribed pleuritis then follows. If any of the bronchial tubes open into the abscess there will also be pneumothorax, and it may happen that the pleuritic effusion will be ejected through the air-passages,—a phenomena which however occurs much more frequently as a consequence of a reverse succession of the processes, namely, from primary pleurisy and consecutive corrosion and suppuration of the pleura. (See Pleuritis.) Finally, pulmonary gangrene sometimes arises in its vicinity, and the purulent solution of the tissues is converted into gangrenous ichor.

When the abscess has existed for a long time, its inner wall appears smooth, and its form is as nearly as possible round, and in the surrounding parts a secondary, interstitial inflammation may be observed, in consequence of which the parenchyma becomes converted into a cellulo-fibrous tissue, which surrounds the cavity of the abscess, and isolates it from the remainder of the pulmonary tissue. When an abscess is large, its perfect closure is very difficult; the process by which this is effected is by agglutination of its walls, which causes the obliteration of the bronchi entering into it; when the abscess has been a very large one, there is a depression of the thorax over it; and when its position is near the surface of the lungs, there is a puckered cicatrix left.

A pulmonary abscess may be confounded with a tuberculous vomica, and with certain accumulations of pus, which are developed from a secondary inflammation of the capillaries of the pulmonary tissue, of which we shall speak presently, and also with saccular dilatation of the bronchi.

The diagnosis may be established from a comparative view of the positive signs attending each of their conditions.

Induration.—There are certain conditions under which hepatization does not pass into a state of purulent solution, but into induration. The red inflammatory product becomes of a grayish-red tint, and finally gray, but instead of becoming dissolved, it becomes compact and indurated. This is what has been termed *indurated hepatization*, a condition which has sometimes, but incorrectly, been regarded as chronic pneumonia. The lung is compact, but fragile and pale, and has lost some of the increased size which it has attained during the stage of red hepatization; it still, however, retains its granular texture, which even becomes more obvious, in consequence of the granulations becoming more marked owing to their increased density, although they are somewhat smaller.

This condition may exist for a long time, and is always followed by cachexia, and especially by dropsical symptoms, and it often proves fatal; or the induration may be gradually resolved, or merge into obliteration of the air-cells and atrophy of the tissue.

The curative process in indurated hepatization is somewhat analogous to the resolution of the pneumonia in the second stage, for an exhalation of serous fluid takes place from the inner wall of the air-cells and acts as a menstruum, which gradually corrodes and absorbs the indurated granulations. As the granulations become smaller it becomes turbid and flocculent, and when the pulmonary cells are again permeable to air, it gradually assumes a frothy appearance.

In other cases the air-cells contract over the granulations, coalesce with them round their circumference, and become obliterated, their tissue being changed into a cellulo-fibrous structure, in which from the similarity of their organizations, the granulations are most probably also merged. Unless a serous effusion occupy the empty space, this termination causes a depression of the thorax, or bronchial dilatation, or both simultaneously; and it appears to be on the whole less frequently the result of the croupous pneumonia which has already been described than of an insidious inflammation of the interstitial tissue,—interstitial pneumonia.

The above is a sketch of croupous pneumonia in general, when it occurs as a *primary* disease; but it also very frequently occurs as a *secondary* process. It most commonly runs an *acute* course, usually passing through its different stages in from two to three weeks, and in extremely rapid cases even in three or four days; these are, however, of rare occurrence. Sometimes, on the other hand, it runs a *chronic* course, being either nearly uniformly prolonged in all its stages, or one or other of them being especially protracted. It presents, however, no special relations essentially different from the sketch we have already given; for, like the acute form, it usually ends in purulent infiltration, and rarely in abscess or induration; and it is totally different from the affection which we commonly find described in pathological treatises as chronic inflammation of the lungs, and with which we shall become acquainted when treating of inflammation of the interstitial tissue.

We observe variations in regard to the original *extension* of pneumonia, which are of importance chiefly in consequence of their connection with the inner nature of the disease.

Pneumonia, according to its variety, attacks, as we have already described, the whole of one of the larger divisions of the lung, that is to say, a whole lobe, or a great part of one, and it is then termed *lobar*. It often attacks a whole lobe, and extends to the adjacent ones, and does not prove fatal till at length the remaining healthy lobes begin to be affected. It usually appears in this form as a primary affection; its most common seat is in the lower lobes, and the right lung is more frequently attacked than the left; both these rules present, however, many exceptions.

Or it attacks only smaller portions of the lungs, a number of individual lobules or single aggregations of lobules, between which we find the parenchyma in a comparatively normal state. It is then termed *lobular pneumonia;* it must be distinguished from the lobular hepatizations which are produced by irregularity in the progress of a lobar pneumonia in the individual lobules, while the rest of the parenchyma remains in a state of inflammatory engorgement.

Or, finally, the seat of pneumonia is confined to single air-cells; we then have what is termed *vesicular pneumonia.* The disease passes through the stages of inflammatory engorgement, of hepatization, and of purulent infiltration in a single air-cell, or it causes induration, and, finally, obliteration of it. The indurated hepatization of single air-cells is undoubtedly the same condition that has been described by writers on pathological anatomy, as *Bayle's pulmonary granulations*, and regarding whose nature there has been much unnecessary dispute. It is undoubtedly the result of inflammation, and so far Andral is correct in his view; but inasmuch as the inflammatory product, under certain conditions, assumes the character of tuberculous matter, it may also be regarded as partaking of the nature of tubercle (Laennec and Louis). It represents, as we shall presently show, the tuberculous infiltration of single air-cells.

Lobular and vesicular pneumonias are usually secondary processes.

It is very important that we should understand the differences presented by the inflammatory product in regard to its plasticity, inasmuch as they are most intimately associated with the condition of the blood (the general disease). Instead of the plastic, hepatized product, we meet under various conditions with serous, flocculent, and turbid, or gelatinous and glutinous or sero-purulent, or even ichorous infiltrations, which, in consequence of their deficiency in coagulable matters, can never give rise to a granular texture of the parenchyma (hepatization). The lung adjacent to the infiltration is dense and spleen-like, and in addition to the other marks of the inflammatory stasis, is generally discolored, somewhat resistant to the touch, but on closer examination is found to be yielding, and is easily torn. Primary acute pneumonia usually deposits a plastic, hepatizing product, which goes through the metamorphoses which have already been described; while, on the other hand, the last-named products are often the result of sluggish, asthenic (hypostatic) inflammations, and even more frequently of secondary pneumonic processes; they represent secondary exudative processes which not unfrequently degenerate into gangrene.

One of these pneumoniac infiltrations, namely the *gelatinous*, must here be especially noticed. It is altogether different from the condition to which Laennec applied the denomination of gelatinous tuberculous infiltration, and which Andral, without hesitation, put down as the product of inflammation, and which we regard as the product of an inflammation of the interstitial tissue (see p. 78). In place of the plastic hepatizing product, the air-cells are found to contain a gelatinous, viscid fluid, sometimes almost resembling frogs' spawn, and of a grayish, grayish-yellow, grayish-red, or brownish-red color, and either clear and transparent, or flocculent and turbid, while the parenchyma is of a pale red tint, or more frequently of a reddish-brown color, and is easily torn. The pneumonia which deposits this non-plastic product is chiefly observed around pulmonary tubercles, and especially around infiltrated tubercles and hepatizations which are undergoing metamorphosis into tuberculous infiltration; it is developed towards the end of the disease, and sometimes involves all the parenchyma which had remained free from tubercle and tuberculous infiltration; moreover we sometimes observe it in the vicinity of extensive hepatization, especially on the border of a hepatized lung in which emphysema has been developed, and which is impervious to a dense injection. Finally, it occurs whenever there is a deficiency of plastic matter for the deposition of a coagulable, hepatizing product, either from some primary cause or in consequence of too profuse previous exudations.

Finally, we must here offer a few remarks on certain metamorphoses which constitute a hitherto undescribed termination of pneumonia, and which the plastic (fibrinous) hepatizing product of inflammation undergoes in consequence of an inherent peculiar constitution depending on a general dyscrasia. These are its very frequent conversion into tubercle in the form of *tuberculous infiltration* or of *infiltrated tubercle*, and its very rare transformation (organization) into medullary cancer, as *cancerous infiltration* or *infiltrated cancer of the lung*, to which we shall again return in our remarks on tuberculosis and cancer of the lungs.

There is a peculiar form of pneumonia to which the term *hypostatic* has been given by Piorry, and which is developed from the passive stasis which occurs in the most dependent parts of the lung, and to which the term *pulmonary hypostasis* has been assigned (see p. 59). It presents the thorough stamp of asthenic inflammation; for it is usually inert in its course, and lingers for a prolonged time in the stage of stasis, the parenchyma being of a dark livid color, and gradually developing from isolated spots a lax, soft, livid-brown hepatization, which may either be general or limited to several foci, while a considerable portion, and occasionally even the whole, may become the seat of an inflammatory product in the form of a sero-purulent or gelatino-purulent infiltration without a trace of hepatization. It constitutes the foundation of most of what are called latent inflammations of the lungs.

Primary pneumonia especially attacks vigorous adults, although delicate persons are also liable to this disease, and, indeed, not unfrequently seem decidedly predisposed to it; up to advanced age it is generally lobar, attacking at the least the whole of a lobe, and depositing a plastic, hepatizing product in it. It further occurs in children, and even in newborn infants, presenting in this case several peculiarities; the granular texture of the hepatized lung is generally only indistinctly seen, owing most probably to the density of the organ and the smallness of its cells;

moreover the termination in abscess is relatively more frequent in children than in adults, and the lobular form is more frequently met with at this early age, although the simple catarrhal pneumonia is often mistaken for it. It arises in consequence of the influence of a peculiar atmospheric condition which predisposes to inflammation accompanied with abundant plastic exudations, and it may then be excited by many even very trivial causes; and in this point of view a notice of the combinations into which the primary pneumonic process enters is of importance, since they proceed from a common primary cause, namely a peculiar, spontaneous, morbid change in the blood.

One of the constant symptoms is the sympathetic affection of the visceral surface of the pleura of the inflamed lobe, in the form of a thin plastic exudation investing it.

The anomalous condition of the blood which occurs in the pneumonic process, as well as in the other primary exudative processes, is a subject of much importance, since in this affection the change occurs in the most marked form and in the highest degree. In consequence of this circumstance we always find fibrinous coagula in the cavities of the heart as well as in the large vessels and their branches, and not unfrequently in those ramifications of the pulmonary artery which supply the inflamed lobe; they are distinguished by their yellowish and greenish color, by their firmness, by a more or less decided metamorphosis into pus in their interior, by their similarity to the exudations on membranous expansions, and by their being woven among the trabeculæ of the heart; and their partial coalescence with the endocardium and the inner membrane of the vessels, together with an obvious appearance of a secondary irritation in them, combine to show that they, at least in part, originated during life.

Pneumonia, if we except the pleurisy which coexists with it, very frequently occurs as an independent disease (an exudative process) upon an extensive surface of mucous membrane, and may become more widely diffused in the lobar form, although it may, on the other hand, often be combined with similar processes upon other structures. Of these processes croup in the final ramifications of the bronchi is far the most common (Lobstein), and is indicated by the presence of creamy, purulent dissolving coagula in them. In children it occurs in combination with croup on the tracheal and other mucous membranes, and with exudations on serous membranes, as pleuritis, pericarditis, meningitis, &c.

Much interest attaches to the combination of pneumonia with secondary inflammations of the lining membrane of the blood-vessels, such as arise either from spontaneous coagulation of the fibrin in high degrees of hæmitis, and its becoming dissolved into pus, or, above all, such as occur in inflammations of the spleen terminating in ulcerous splenic phthisis.

Primary pneumonia proves fatal by inducing paralysis of the lungs; also from the supervention of pulmonary œdema or of other complications, from the high degree of blood-disease and the occurrence of spontaneous coagulations in the heart and vessels, and from acute softening of the stomach and œsophagus.

Secondary pneumonia is frequently developed as a result of inflammations in other organs, when they cause the blood to assume a consecutive disease similar to the spontaneous affection which we have already noticed; it frequently also accompanies specific processes which in their nature are allied to the exudative, and hence it especially occurs in the acute exanthemata. In both these cases the pneumonia is usually *lobar*. Finally, secondary pneumonia may occur as a metastasis towards the termination of various forms of acute dyscrasia of the blood, which, in their course, degenerate into a croupous diathesis; amongst these we must place many exanthematous, and the typhous and tuberculous processes; the pneumonia in these cases is generally *lobular* and may even be *vesicular*. Under this class we may also place many of the so-called latent, symptomatic, and, as has been already remarked, the metastatic inflammations. They are combined, especially under the circumstances which we have just mentioned, with exudative (croupous) processes of various degrees of plasticity on other mucous and serous membranes.

From all that has been stated, the croupous nature of the pneumonic process in general is sufficiently clear; being always based either on a peculiar primary (spontaneous) or on a secondary disease of the blood. There can be no doubt that this condition constitutes the basis both of secondary pneumonia and of the other metastatic croupous processes so frequently combined with it, and it may also, in all essential points, be looked upon as the foundation of primary pneumonia and other primary croupous processes on the mucous membrane of the mouth, throat, and respiratory organs.

But as there are variations in the individual peculiarities, the age, and the external influences, under which croup of the mouth and pharynx, tracheal and bronchial croup, and, finally, croupous pneumonia are developed, so also may the diseased condition of the blood vary in these affections, although probably only in a slight degree; and pneumonia, if we consider it as pulmonary croup, and if we take into consideration the plasticity of the exudation, may be regarded as occupying, in adult life, the same place which in earlier life is held by pharyngeal and tracheal croup; while bronchial croup, especially in adults, forms the transition between the two latter varieties and pulmonary croup (croupous pneumonia). We now proceed to the consideration of *typhous pneumonia*, in consequence of the similarity of its anatomical relations to those of croupous pneumonia.

Typhus Pneumonia (*Pneumotyphus*).—The pneumonic process is very frequently associated with the typhous; but its relation to the latter, and especially to the local typhous process on the mucous membrane of the ileum, is not always the same, and hence its importance varies.

In all cases of typhus, and especially when there is well-marked ileotyphus, there is hypostasis in the lower lobes; and this not unfrequently becomes developed into pneumonia, which deposits a gelatinous, glutinous, soft product, similar to the typhous, bronchial, and intestinal secretion, and corresponding to the existing typhous dyscrasia. It is the result of an adynamic state of the system, and bears no further definite relation to the typhous process, which is seated on the intestinal mucous membrane.

A more intimate relation, however, exists when the typhous process has been originally localized in the pulmonary mucous membrane to the exclusion of other structures, especially the intestinal mucous membrane, namely, in *primary pneumotyphus*; and when, in consequence of its absolute intensity or its relatively imperfect localization on the intestinal mucous membrane, it also appears in the lungs, and completes the local process on the intestinal mucous membrane, as *secondary pneumotyphus*.

Primary Pneumotyphus is a (croupous) lobar pneumonia characterized by the livid and almost violet color of the parenchyma during the first stage, and by a dirty brownish-red or chocolate-colored, very yielding inflammatory product (hepatization), which soon breaks down when there is great disease of the blood, and extreme absence of plasticity. It seems to be always combined with bronchial typhus, and the bronchial glands exhibit the characteristic relations of this affection. It exists either without or with only a slightly marked secondary affection of the intestinal mucous membrane, and, in association with bronchial typhus, doubtless constitutes most, if not all, of those cases of typhus—and especially exanthematous typhus—which run their course without any local intestinal affection. Like genuine pneumonia it is usually combined with the pleurisy yielding a similar product.

Secondary Pneumotyphus in its genuine form consists of an imperfectly developed local typhous process on the intestinal mucous membrane, has the same anatomical characters, but does not, as a general rule, attain the same degree of intensity and extent, which is presented by the primary form when it meets with no obstruction to its original local development. It also enters into the same combinations, and is very frequently associated with genuine secondary laryngotyphus.

Secondary pneumotyphus occurs, however, much more frequently in a *degenerate* form, as a local expression of the degeneration of the collective typhous process, and, indeed, in the form of a lobular or vesicular pneumonia yielding a purulent and diffluent product, and very frequently associated with a form of laryngotyphus which has degenerated into croup; or it occurs in the form of purulent, diffluent deposits in the interstitial tissue, with inflammation of the capillaries of the lungs (purulent metastasis); or finally in the form of pulmonary gangrene.

2. *Catarrhal Pneumonia.*—Catarrhal pneumonia has hitherto received little attention, in consequence of its resemblance to the croupous variety, for which it may easily be mistaken, and on account of its rare occurrence in adult life. It is, however, comparatively common in children, in consequence of the large amount of undeveloped granular texture that is observed in hepatization of their lungs.

It constitutes the first of the series of catarrhal affections to which the respiratory mucous membrane is exposed during childhood, and is succeeded in later years by bronchial and, finally, by tracheal catarrh: in this respect it is the opposite to croup, which begins in childhood as pharyngeal and tracheal croup, and which, in the form of pulmonary croup, terminates the series of croupous inflammations in adults. Catarrhal pneumonia is always lobular, and associated with a catarrhal affection of the bronchial tubes pertaining to the diseased lobules; it is frequently found in the various catarrhal affections of children, especially in pertussis and catarrhus suffocativus. Its usual position is in the superficial lobules, of which a very considerable number are often affected. They present, for the most part, a bluish-red tint, and are dense and somewhat firm; the walls of their air-cells are swollen, till no internal cavity remains, or if the swelling be less considerable, their cavities contain a watery, mucous, and slightly frothy secretion; there is no trace of granular structure. As the lung-substance in the immediate vicinity of the diseased lobules is usually emphysematous and pale, they appear to be a little depressed below the level of the surrounding lung if they are situated near the surface, and they may be further recognized by their dark color.

This disease frequently becomes fatal by the supervention of pulmonary œdema and paralysis, or by the stasis induced in the heart by the emphysema.

3. *Inflammation of the Interstitial Tissue of the Lungs. Interstitial Pneumonia.*—This is a disease whose anatomical characters are not properly recognized in pathological treatises, for it is commonly described as *chronic inflammation* of the lungs consequent on ordinary croupous pneumonia, without any reference to its seat in a special tissue. The seat of this inflammation is the interstitial cellular tissue of the lungs, although the walls of the air-cells are also implicated, in which case the pneumonia sometimes assumes the croupous form.

Its course is, as a general rule, chronic, and it is only very rarely that we have the opportunity of studying it, except in its final effects. So far as we can conclude from our few observations, it appears to commence in the tissue lying in the interstices of the pulmonary lobules and between the smaller groups of air-cells, which, if too much black lung-substance be not present, becomes of a pale red color, and is swollen by albuminous infiltration, while the air-cells are either pale and more or less compressed in proportion to the swelling; or, if they are involved in the inflammation, they appear reddened and, in accordance with what has been already stated, sometimes finely granular. In the progress of time the infiltration within the interstitial tissue becomes organized and coalesces with the latter, so as to form a dense cellulo-fibrous substance, which compresses and obliterates the air-cells, and finally converts them into a similar cellular tissue. We then find either whitish, hard stripes, which not unfrequently grate under the knife, or irregular masses interwoven in the lung-substance.

This is the ordinary metamorphosis consequent on chronic pneumonia; in some cases, however, it may terminate in suppuration which isolates the individual lobules; and some pulmonary abscesses probably originate in this manner.

It is not very frequently a spontaneous affection, insidiously spreading from one lobule to another; it is commonly seated in the apices of the upper lobes, and as we may infer from the coexisting cellular adhesions corresponding to their seat and distribution, it is frequently combined with circumscribed pleurisy.

The affected portions of the lung become depressed, and draw down the surrounding parenchyma in the form of cicatrix-like folds, which may sometimes be observed on the apices of the lungs in cases where there is no trace of the pre-existence of the tubercle. A further consequence of this process is a depression of the thorax at the corresponding spot, and, internally, a dilatation of the bronchial tubes.

More frequently, however, it is a consecutive affection, arising from reaction, and leading to the production of cyst-like formations around the seat of old apoplexies, abscesses, tuberculous caverns, gangrene, &c.; its products then resemble the tissue of which cicatrices are composed.

This tissue sometimes contains a considerable quantity of pigment (the black pigment of the lungs); it then presents blackish-gray stripes and spots, or else is uniformly of a blackish-blue tint.

g. Deposits in the Lungs. Metastatic Processes.—As a consequence of the absorption of a pseudoplastic process into the living blood, or, more rarely, as a consequence of the spontaneous disease of that fluid, there is a process developed which is fully discussed in its general bearings under the head of "the diseases of the blood:" it, however, affects the lungs more frequently than any other organ, and usually occurs simultaneously at several circumscribed spots. It consists in the deposition of a fibrinous product in the lung-substance, or of a coagulation in its capillaries (phlebitis capillaris), either of which undergoes metamorphoses corresponding to the principle taken up into the blood.

As, on the one hand, the veins seem to be the seat in which deleterious substances are produced, or in which they are collected from without, and as, on the other, the whole of the venous blood passes through the lungs,—the principal organ in the process of hæmatosis,—it is easy to understand why it is that in general these deposits are most frequent and most abundant in the lungs.

As is generally the case in all parenchymatous organs, these deposits almost always occur in the superficial layers of the lungs. We find deposits of various dimensions, from the size of a millet-seed to that of a lentil, a pea, a bean, or even a nut, scattered through the tissue of the lung, and separated from one another by large patches of healthy tissue; the smaller they are, the more they resemble, in form, a roundish granulation, while on the other hand, the larger they are, the more they lose the round form, and appear as irregular, angular, ramifying masses. Large deposits, when lying near the surface, and pressing upon the pulmonary pleura, like those occurring in the spleen, have a wedge-like shape, being thick externally, and growing small towards the interior. They are at first of a blackish-red or brownish-red color, and firm although fragile, and can be distinguished by their sharply defined outline, and by their apparently homogeneous structure, from the surrounding tissue, which at the commencement is normal, or at most the seat of hyperæmia and œdematous infiltration; but subsequently, when the deposit begins its progressive metamorphoses, a reactive inflammation, in the form of croupous pneumonia and hepatization, is set up in the lungs, and its extent is usually proportioned to the size of the deposit.

The deposit subsequently becomes of a lighter color, and undergoes one of the following metamorphoses:

In one case (and this is what commonly occurs), the deposited mass becomes more or less decolorized, and dissolves into a cream-like, purulent, or ichorous fluid, which destroys the tissues. This process commences in the centre of the deposit, much as we observe to take place in secondary phlebitis of one of the larger veins, and we then find the above-named fluid enclosed within the outer remains of the deposit, around which a reactive inflammation is established. In the course of time these, and the adjacent tissue also, undergo a similar process of fusion, and the extent of this change is proportional to the destructive tendency of the product of the reactive inflammation. Moreover, this process is very often essentially of a septic nature, and is based on the absorption of gangrenous ichor, or, on the other hand, it often undergoes degeneration, and gives rise to gangrene of the surrounding tissues. These deposits are very frequently combined, from the first, with a secondary pleurisy of a croupous nature; sometimes, however, the latter occurs as a consecutive affection, arising from the inflammatory reaction that is set up around the superficial deposits, or as a purulent or ichorous abscess in the immediate vicinity of the pleura. In the latter case, we observe the abscess as roundish, nodular, furuncular, yellow prominences, or if gangrenous destruction has occurred, as dirty greenish or brownish collapsed spots, shining through the pulmonary pleura, which itself undergoes destruction from suppuration or gangrene, with or without perforation, and gives rise to general pleurisy.

In the other metamorphosis, which, however, is extremely rare, the deposit, without dissolving or undergoing any intermediate change, passes directly from its crude state into that of obsolescence, that is to say, it shrinks into a callous, grayish nodule, which is seated in a capsule of cellulo-fibrous tissue, and in the course of time becomes converted into an osseous concretion. Many of these peripheral deposits, after their conversion into concretions, have doubtless been mistaken for chalky tubercles. The more complicated retrograde process which is sometimes manifested in deposits in other parenchymatous organs, as for instance the spleen and the kidneys, namely, the cheesy disintegration of the product, and its subsequent conversion into chalky matter, may, as we should presume from analogy, also occur in the lungs; but in the whole course of our observations, we cannot recollect a single case in which it has occurred.

We have spoken in the first volume of the rarity of obsolete deposits in the lungs, and have also accounted for it.

We have already explained how these deposits become combined with pleurisy; they are also associated with similar deposits in other parenchymatous structures—as the spleen, kidneys, liver, brain, and thyroid gland, in the tissue of mucous membranes, especially that of the intestines, in the skin, the subcutaneous cellular (areolar) substance, and all interstitial cellular layers, and in the muscles; also in the exudative processes on mucous, serous, and synovial membranes (as, for instance, metastases in the joints).

They must be carefully distinguished from lobular pneumonia, for which they have sometimes been mistaken.

h. Gangrene of the Lungs is an affection of not unfrequent occurrence, and one which, as Laennec very correctly remarks, must not be regarded as the result of an excessively acute inflammation. We do not, however, intend to assert, that it cannot by any possibility occur in an inflamed lung, for under certain conditions hepatization of a portion of the lung is unquestionably the most common complication.

We will first consider it in an anatomical point of view, and then proceed to notice the conditions under which it is developed.

There are two perfectly distinct forms of gangrene of the lungs, namely, *diffuse* gangrene and *circumscribed* gangrene or *gangrenous eschar.*

In *diffuse* gangrene, we find a portion of the lung presenting an abnormal greenish or brownish tint, filled with a similarly colored, somewhat frothy, turbid serosity, soft, rotten, and readily breaking down into a pulpy, shaggy tissue. The whole evolves the characteristic odor of sphacelus. Towards the outer portion the discoloration, infiltration, and alteration of consistence are less marked, and finally become imperceptible; and there is no line of demarcation between the gangrenous and the adjacent tissue, which only differs from the normal state in being œdematous and anæmic. It corresponds to diffuse gangrene of the bronchial mucous membrane, with which it is almost always associated. Upon the whole it is a rare affection; but when it does occur, it always attains a considerable extent, as it commonly attacks the whole of a lobe, or, at all events, its greater part. It especially attacks the upper lobes, when, in consequence of excessive activity, they have become the seat of emphysema and anæmia, the lower lobe being at the same time in a state of passive stasis. It is perhaps scarcely entitled to rank as an essentially independent affection, inasmuch as it is almost always associated with gangrenous eschar of the lungs; and hence it is the more readily developed from the contact of the ichorous, gaseous, and fluid products of the gangrenous eschar coming in contact with the bronchial and pulmonary mucous membrane, inasmuch as in all probability the disease extends from the bronchi to the lung-tissue. The above description of gangrene, as it occurs in the upper lobes, is sufficient to render this form intelligible, as well as to explain why there is no inflammatory reaction, and consequently no line of demarcation around the affected tissue.

As we have already remarked, it must be carefully distinguished from *softening of the lungs.*

Circumscribed or *partial Gangrene* of the lungs appears in the form of gangrenous eschar, and is incomparably more frequent than the former variety. We find the parenchyma, at some spot of varying size, converted into a blackish or brownish-green, hardish, but moist and tough eschar, which adheres to the surrounding tissue, evolves, in a very marked degree, the peculiar odor of sphacelus, and, as Laennec observes, is extremely similar to the eschar produced on the skin by nitrate of silver. It is sharply defined, and, as we shall presently show, the surrounding parenchyma may be in various conditions.

The eschar becomes gradually loosened from the surrounding tissue, and rests in an excavation corresponding to it in size and form; it may be described as a blackish-green plug, which superficially is soft, shaggy, moist, and bathed in an ichorous fluid, but, towards its centre, is of a denser structure. More frequently, however, the whole or the greater portion of the eschar softens and becomes dissolved into a greenish-brown, very fetid, ichorous pulp, mixed with rotten, shaggy fragments of tissue, and enclosed in a cavity whose walls are lined by a shaggy tissue infiltrated with ichor.

The size of the gangrenous portion, at its commencement, varies from that of a bean to that of a hen's egg, or may be even larger; it is most commonly not smaller than a hazel-nut or larger than a walnut. The form is on the whole irregular, with a tendency to roundness. It is much more commonly seen in the superficial than in the deep layers of the lung, and more frequently in the lower than the upper lobes.

These eschars may either occur singly, or several may be simultaneously present.

The number and size of the bronchial tubes attacked by the gangrenous destruction, are usually proportional to the size of the original gangrenous centre; these bronchial tubes constitute the passage through which the gangrenous exhalation and the eschar itself, in the respective form of an intolerably fetid atmosphere around the patient, and of gangrenous, ichorous sputa, make their escape. The gangrene proceeds outwards, and attacks the pulmonary pleura the more quickly the nearer it was originally seated to the surface of the lung. If the gangrenous eschar becomes detached, it falls into the cavity of the thorax, unless there are firm adhesions at the spot; or else it becomes dissolved, and the ichorous semi-solid matter is effused into the pleural sac, and gives rise to pleurisy with ichorous exudation, and to pneumothorax, since the fetid gas evolved from the gangrenous mass either collects alone in the thorax, or atmospheric air finds its way through the bronchial tubes which open into the abscess, and thus mixes with the aforesaid gas in the thorax. These superficial gangrenous caverns may be recognized at a glance, for at these spots the pleura is either converted into a blackish-green eschar, whose inner surface is shrivelled and hard; or, if the eschar has already dissolved, the pleura is of a blackish-green color, rotten, and moist, and appears distended by the gas evolved from the abscess; or, finally, if the pulmonary pleura be ruptured at certain spots, or be perforated, or even perfectly destroyed, in consequence of spontaneous fusion, we shall observe the open, sunk cavern, either covered by the remains of the pleura, or thoroughly exposed, and more or less completely emptied.

A primary gangrenous abscess must be distinguished, when possible, from one that has undergone subsequent enlargement; very large abscesses are, as a general rule, not primary, but are formed by the corroding action of circumscribed gangrene, and do not, as we shall presently show, present the distinct line of demarcation which is observable in primary abscesses.

The lung-substance surrounding the gangrenous abscess is sometimes normal, with the exception of a serous or sanguineo-serous infiltration; but when the gangrenous eschar dissolves, diffuse gangrene may be developed in it to a greater or less extent. More frequently, however, we see it in a state of reactive inflammation, varying in extent and character. Very often there is a simple stasis of an asthenic character; this gradually assumes an inflammatory type, which it retains for a long time, and then the stage of hepatization slowly and imperceptibly ensues. From a want of energy in this process of reaction, the primary gangrenous abscess may extend in various directions, so as often to attain the size of a man's fist, or even of a child's head, while the surrounding tissue becomes more or less rapidly discolored, without presenting any decided stratified appearance, and finally breaks down into a gangrenous, ichorous pulp. In this way the gangrene may extend outwards, until it reaches the pulmonary pleura, when it may give rise to the consequences which have been already enumerated; and indeed, if the lung be adherent, the costal pleura at the corresponding spot may be involved in the metamorphosis.

We often find a higher degree of inflammation set up in the surrounding tissue; it is in a state of decided hepatization, which sometimes extends over the whole of the lobe which is affected by gangrene. The disease not unfrequently proves fatal through this excessive reaction.

The most important process, however, occurs in the layer of tissue immediately surrounding the cavern, and is obviously an effort of nature to promote a cure. The reaction here appears as an inflammation of the interstitial tissue of the lungs, which, together with the cavernous walls, undergoes suppuration, and thus effects the removal of the sphacelated tissue which was adhering to the walls of the abscess.

In this process we find that at first only single or isolated patches of tissue become gangrenous, and the pus which is secreted from the walls of the abscess is still mixed with ichor, and gangrenous fragments of tissue. As the process advances, however, suppuration predominates, and after the gangrenous tissue has been ejected through the bronchi the cavern is converted into an ordinary suppurating abscess, whose inner wall is infiltrated with pus; externally, for a distance varying from three to six lines, the tissue is of a grayish-red color and firm; and if croupous exudation in the air-cells be associated with the inflammation of the interstitial tissue, we observe a scarcely perceptible, very delicate granulation. If the suppuration in the inner stratum of the capsule now diminishes, the result of the whole process is a cavity, with whitish, cellulo-fibrous, callous walls, which sooner or later coalesce, leaving merely a cicatrix, like ordinary abscesses or tuberculous vomicæ. In some rare cases circumscribed pulmonary gangrene undergoes a cure in this manner.

If the eschar breaks down, and dissolves very rapidly, and little or no reaction be developed in the surrounding parts, or if the primary cavern enlarge very quickly at the expense of the surrounding parts, the gangrenous destruction not unfrequently involves large, unobliterated blood-vessels, and gives rise to exhausting hæmorrhages into the cavern, the bronchial tubes, or even into the thoracic cavity, when the abscess has opened into the pleural sac.

Partial gangrene often arises in the perfectly healthy lungs of weak, decrepit, cachectic persons from general depressing influences, and is developed from a circumscribed passive stasis. Under similar circumstances, we find it associated with pneumonia in its various stages, with pulmonary abscess, with pulmonary tuberculosis and tuberculous vomicæ, with bronchitis, especially when it is developed in the course of exanthematous diseases, both in adults and children, &c. Finally, it appears among the sequelæ of typhus, as a manifestation that the typhous process is spontaneously degenerating into a state of putrescence; or it may be produced by the absorption of gangrenous ichor from gangrene of different parts into the blood, in which case we have diffluent gangrenous deposits, or septic capillary phlebitis.

i. Softening.—Softening of the lung-tissue is of very rare occurrence; it is altogether distinct from pneumonia, and must not be confounded with Andral's ramollissement (red and gray hepatization); like softening of the stomach, it is a peculiar spontaneous process, and appears under precisely the same conditions as that affection: indeed, as a further proof of their identity, we may add that this disease is almost always combined with gastric softening.

In any part of the lungs we may find an undefined patch of a dirty brown or blackish color, according to the state of the blood at the commencement of the process, and which is so very moist and soft that on the slightest pressure it breaks down into a pulp, which is mixed with a serous fluid and contains black flocculi of carbonized blood. The bronchial mucous membrane is found in the same state for some distance around the diseased spot.

In consequence of the considerable quantity of blood which is always contained in the lungs, there is a resemblance between softening of the pulmonary tissue and the black softening of the stomach, which proceeds from a disease of the blood itself. It may be easily mistaken for diffuse pulmonary gangrene; indeed in very intense cases the diagnosis must depend on the absence of the gangrenous odor, and on the lesser degree of discoloration.

k. Adventitious Products.

1. *Cysts* are of extremely rare occurrence in the lungs, which in this respect present a marked contrast with many of the other parenchymatous organs.

Simple Serous Cysts may, doubtless, occasionally be found in the lungs, but *sacs containing Acephalocysts* are of less rare occurrence. The rarity of the latter cysts in the lungs contrasts strongly with their frequency in the liver, and this is very important when we consider the frequency of pulmonary tuberculosis, for this, in addition to the inverse ratio of the frequency of these secondary products in other organs, especially in the liver, constitutes one of the most important objections to the theory that tubercle has a hydatid origin.

Hitherto only single sacs of acephalocysts appear to have been found in the pulmonary tissue; they have varied from the size of a pigeon's egg to that of a man's fist, and have occurred sometimes in the upper and sometimes in the lower lobes.

They are undoubtedly developed in the interstitial tissue of the lungs, and occasion, according to their size, more or less compression of the parenchyma, which is thus gradually converted into fibro-cellular tissue (obsolescence). The parent sac is surrounded by and adherent to this tissue, and contains, in its interior, the acephalocysts, which vary in number and form, and either swim freely in a serous fluid or are attached to the walls.

It is important to recollect that in rare cases the parent sac may be destroyed by inflammation and consequent suppuration, and a communication may thus be established between the cavity and the bronchi, through which the acephalocysts may be ejected, especially as in less rare instances acephalocysts are ejected from the liver by this complicated route.

The pulmonary sac containing acephalocysts often communicates with a similar sac in the liver.

Cysts containing other substances, as for instance cholesterin, with or without hair, are even rarer than cysts with serous contents.

2. *Anomalous Fibrous or Fibrocartilaginous Tissue* occurs—

a. As *callous condensation* arising from chronic inflammation of the interstitial tissue; it likewise occurs as *cicatrix-callus* around old abscesses, tuberculous cavities, apoplectic effusions, &c.

b. Fibroid Tumors are incomparably rarer. They never attain any considerable size, being seldom larger than a bean or a hazel-nut. They are either bluish-white, firm, elastic, very dense and flat bodies, or, as is more frequently the case, they are of a pale yellow or dirty white color, flabby, soft, and puckered, and resemble the structure of the mammary or salivary glands.

3. *Anomalous Osseous Substance* occurs not only in the bronchi (see p. 35) and bronchial glands, but under various circumstances, in the lungs, especially in the form of ossification of anomalous fibrous tissue or of the chalky metamorphosis of an unorganized structure. To the first belong many either flat or roundish and nodular, yellow, and generally very compact concretions, which are developed in and from all the forms of anomalous fibrous tissue, but especially in the callous stripes, capsules and cicatrices; to the latter belong the chalky, whitish or grayish nodular, brittle, and even friable masses into which tubercle and tuberculous pus are, under certain conditions, metamorphosed.

4. *Black Pigment* is more frequently and abundantly deposited in the lungs and bronchial glands than in any other organ, except the mucous membrane of the intestinal tract. It occurs, with rare exceptions, in the lungs of all adults, and increases with advancing years. Hence it can properly be only regarded as a pathological appearance, either when it occurs in the earlier periods of life or in excessive quantity.

The pigment which occurs in the form of molecules is either deposited in a *free* state in the interstitial tissue and in the walls of the air-cells, or else it is *combined*, as a new formation, with some older deposit.

In the *first* case it is found according to the extent of the accumulation, in blackish-gray, blackish-blue, or ink-black points, or in patches, as if laid on with a brush; or if very abundantly present, it is diffused over the interstitial tissue in large ramifying streaks, which appear as islands in the cellular tissue under the pulmonary pleura, and are uniformly infiltrated, blackened, and as it were inked, and are thickened and tough. This thickening of the interstitial cellular tissue is important, since it impedes the development of the air-cells, and likewise gradually obliterates their vessels, and in this manner causes their atrophy. We must here especially notice a metamorphosis which not unfrequently occurs at the apices of the upper lobes, and is unassociated with any other anomaly; we refer to the deposition of large quantities of pigment which give a black color to the tissue, and increase its firmness, its structure being either normal or presenting at some spots an irregular reticulated appearance in consequence of atrophy. *Senile atrophy* of the lungs is undoubtedly often induced by an excessive accumulation of pigment in the interstitial tissue. The deposit usually takes place through the whole lung, but is most abundant near the surface and in the upper third of the superior lobes. It is the result of slight irritative processes and of transient stases; the pigment is conveyed by absorption to the bronchial glands, and is thus deposited in them. It must be decided by further and more careful observations whether the larger deposits of pigment which are so frequently noticed in the lungs of persons engaged in working both coal and coal dust, depend upon the actual absorption of these extraneous matters into the tissue, or whether, as we are more inclined to believe, they are the results of the continued irritation to which the pulmonary mucous membrane of such persons is necessarily subjected.

In the *second* case the pigment is the result of a chronic pneumonia, and we find it infiltrated in various quantities into an indurated and callous parenchyma. We meet with it in the vicinity of tuberculous deposits, especially of hemorrhagic tubercle, and in certain cancerous deposits, especially in cancer melanodes.

5. *Tubercle.—Pulmonary tuberculosis*, which is the most frequent of all the tuberculoses, is one of the most common and likewise the most fatal of the diseases of the lungs.

For general information on tubercle and tuberculosis we must refer to what has been stated in the first volume; we shall here endeavor to apply those general principles to a special case, and at the same time shall attempt to elucidate certain points, which, from presenting some peculiarity, require a fuller notice.

Tubercle does not primarily occur in the lungs in the numerous forms which have been described, but only in *two* forms, which are most essentially connected, both in relation to its mode of formation and its seat. To these two forms we apply the respective terms of *interstitial tuberculous granulation* and *infiltrated tubercle* or *tuberculous infiltration.*

a. Interstitial Tuberculous Granulations occur in the pulmonary texture in the form of roundish, originally gray, semi-transparent bodies, varying from the size of a hemp or millet-seed to that of a barleycorn; these minute bodies either occur singly and in an isolated state, or several are collected into a group, or finally they may coalesce and form a large continuous mass. They are seated, as is shown both by special anatomical investigations and by numerous analogies, in the interstitial tissue between the smallest lobules and the air-cells, and on the walls of the cells themselves; that is to say, they are altogether external to the cell-cavities; but by pressure on the cell-wall they sometimes induce a corresponding internal prominence, or, if they be of larger size, they exert such pressure on the walls, that in every group or confluent mass of tubercles we find a number of cells more or less completely obliterated. It is the result of a *chronic* or *acute tuberculous process*, which is accompanied by local congestion or hyperæmia. We have now sufficiently indicated this form of tubercle, but we shall hereafter return more fully to it. For the sake of brevity we shall always name it *tubercle*, or *tuberculous granulation*, and it must be carefully distinguished from the second form, which we shall invariably term *tuberculous infiltration*, or *infiltrated tubercle.*

b. Infiltrated Tubercle, unlike interstitial tubercle, is actually deposited in the cavities of the air-cells. It arises from a more or less extensive croupous pneumonia whose products, under the influence of a tuberculous infiltration, become variously discolored, and converted into yellow tubercle, instead of being absorbed or dissolving into pus. Hence tuberculous infiltration presents the form of *hepatization*, or more strictly speaking is hepatization, induced by a tuberculous product. The pneumonic product, which was at first red and granular, gradually becomes of a paler and grayish-red color with a tinge of yellow, and is dry and fragile; it finally becomes yellow, moist, of a soft, fatty, cheesy character, and sooner or later becomes disintegrated into tuberculous pus. The granular texture, in the mean time, gradually disappears, whilst the tissue forming the air-cells becomes tuberculous, and the diseased portion of lung appears to be actually changed into a connected fatty-cheesy tuberculous mass,—a condition which Lobstein doubtless observed, and mistook for *fatty metamorphosis* of the lung-substance.

This form of tuberculosis may attack *a whole lobe* uniformly, or even *a whole lung*, according to the extent of the local pneumonic process; it is, however, much more frequently confined to one or several larger or smaller separate portions of lung, and very often occurs as a lobular tuberculous infiltration, and in both these cases it is generally sharply defined; finally, it may occur as *vesicular tuberculous* infiltration, in which case it is the same thing as Bayle's *pulmonary granulations*, regarding which there has been much discussion.

It very often attacks the superficial parts of the lungs, as lobar and lobular infiltration, and may then be at once recognized by its external characters, by the pneumonic tendency, and the peculiar color of the diseased portion.

It is always the result of a high degree of tuberculous dyscrasia, and hence it only rarely occurs as primary tuberculosis, but is as a general rule, associated with advanced stages of interstitial tubercle. It gives rise to a form of phthisis which is tumultuous and acute, is accompanied with repeated attacks of pneumonia, and is attended with much pain and distress.

It is especially frequent in young persons and children, and presents an analogy with bronchial tuberculosis, with one of the forms of tuberculosis of the intestinal mucous membrane, with the tuberculous metamorphosis of exudations on serous membranes, &c. It is always combined with a high degree of tuberculosis of the bronchial glands, and very often with tuberculosis of the intestinal mucous membrane.

These are the two principal forms of pulmonary tuberculosis, and all other varieties of tubercle, such, for instance, as depend on physical peculiarities, however important they may individually be, are unimportant in reference to the local process, depending either on different modifications of the general disease or on mere changes in the tuberculous matter.

There are no organs excepting the spleen and serous membranes in which tubercles occur in such great numbers as in the lungs. They appear either as *separate granulations* or several of them are accumulated into *one group*. In the first case each granulation is isolated from the others by an extent of lung-tissue proportional to the number of the tubercles. This takes place either in a comparatively uniform or in an irregular manner; the latter occurring when in one part of the lung we find a large number of tubercles with little intervening parenchyma, and in another a few tubercles interspersed among much healthy tissue. When the tubercles are present in large numbers they become pressed upon one another, and finally coalesce in the form of irregular masses, as may be especially observed in the apices of the lungs, where the disease is usually the most developed. In many of the more common cases we find an uniform increase in the number of tubercles, and a corresponding approximation of them to one another as we advance from the lower portions of the lungs towards their apices.

This accumulation of tubercles into irregular masses, such as occur in the apices of the lungs, which are the usual starting points of pulmonary tuberculosis, and occasionally at other spots, must be carefully distinguished from *the primary development of tubercle in tolerably regular groups.* Under certain local and general conditions which are not yet altogether understood, tubercles are originally deposited at different spots in groups of a roundish form, and of the size of a pea, a bean, or a hazel-nut, or even larger, while around them there are usually other isolated tubercles in greater or less number. In extreme cases of this kind the tubercles are deposited around a central nucleus of pulmonary tissue, from which processes run into the tuberculous groups, dividing them into several compartments.

Pulmonary tubercles originally appear either (1) as the well-known gray, semi-transparent granulations of the size of a millet or hemp-seed, or in many cases of acute tuberculosis as still smaller-sized granules, which are clear, transparent and vesicular; or (2) in high degrees of tuberculous disease, especially when it is running an acute course, they are separated from the blood as yellow tubercle. On a cursory examination they appear almost or quite round; on closer investigation, however, we find that their outlines are not sharply defined, but that delicate prolongations extend from their surface into the surrounding tissue, which according to their size, may enclose one, two or more air-cells. These cells are most commonly obliterated, but not unfrequently appear dilated.

In examining the lungs we not unfrequently meet with extensive, roundish, or irregularly ramifying or lobulated tuberculous masses, which are produced either by the confluence of several tubercles which were originally in the same group, or by the subsequent deposition of tubercles in the same immediate neighborhood. The tissue at these points is completely wasted away, so that nothing but the pigment remains, and the air-cells and extremities of the bronchial tubes are obliterated. These tuberculous masses must be distinguished as carefully as possible from tuberculous infiltration. The whole of the upper lobes are not unfrequently so thickly strewed with tubercles as to present the appearance of having degenerated into a tough, resistent, uniform tuberculous mass.

In the ordinary course of the disease the principal seat of the tubercles is in the upper third or apices of the superior lobes; it is here that they are deposited first and in the greatest quantity, and that they first begin to undergo their ordinary changes. The apices of the lungs must therefore be regarded as the usual starting point of tuberculosis, which gradually extends from thence to the lower portions of the lungs. Exceptions to this rule are, however, not unfrequent; we sometimes meet with tubercles in the apices and others far away from them, even in the lower lobes, or they may even occur in the latter portion while the upper parts are perfectly free from them. In this respect there is a contrast between pulmonary tuberculosis and pneumonia, at all events in a great majority of cases, for pneumonia most commonly commences in and starts from the lower lobes, while tuberculosis has its origin in the upper lobes, and even in their highest parts.

Many attempts have been made to account for the preference which tubercles exhibit for the upper parts of the lungs, but none of them satisfactorily explain it; they are based either on mere hypothesis, or the cause and the effect have been confounded. We confess our ignorance on this point, and can no more explain it than we can account for the preference shown by certain exanthematous and impetigenous affections for particular regions of the general integument.

Pulmonary tubercles pass through the different metamorphoses which are described in the first volume.

1. They very frequently *soften*, and this change gives rise to *tuberculous suppuration of the lungs*, *tuberculous ulcers*, *tuberculous abscess* (*vomica pulmonis tuberculosa*, *caverna tuberculosa*), and *tuberculous phthisis*. The separate, gray tuberculous granulations begin to soften in their centres, which become turbid, opaque, yellowish and cheesy, and finally undergo purulent solution. The groups of tubercles break down at several points simultaneously, corresponding to the different separate tubercles of which they are composed. Hence, in the first case, we have a small *primary tuberculous ulcer;* and in the second case, after the final solution of the whole mass, a much larger one, whose further progress we shall consider in the following remarks.

It is especially important to understand the manner in which the primary tuberculous ulcers *enlarge*, and give rise to such peculiar and extensive destruction of the lungs. This is elucidated by the process which goes on in the tissue surrounding the softened tubercle. The breaking down of tubercles is always followed by a secondary deposition of tubercle in the surrounding parenchyma, the extent of this secondary deposition being proportional to the intensity of the general disease. Moreover these secondary tubercles and the tissue in which they are deposited likewise break down with a rapidity which stands in a direct ratio to the intensity of the dyscrasia, and in this way the tuberculous ulcer becomes enlarged. If this process goes on in so tumultuous a manner as to exhaust the powers of reaction and the supply of organic matter, the ulcer usually extends unequally in various directions, and forms an irregular, sinuous, and apparently lacerated cavern whose walls consist of lung substance plugged up with softened tubercle, and whose internal surface presents, as it were, a gnawed appearance, without a trace of any inner lining except a coating of adherent tuberculous pus. The small quantity of parenchyma occurring between the tubercles is in a state of compression and dirty brown discoloration (carnification), while that in the surrounding neighborhood exhibits no trace of reaction, except a certain degree of hyperæmia. If this, as is usually the case, takes place simultaneously at several spots, two or more caverns will come in contact and will finally unite, and we then have either a number of caverns communicating with one another by means of sinuses of varying width, and either straight or tortuous in their course, or else the whole represents a large abscess with sinuosities in various directions. This cavity is intersected in various directions by bridges or rafters of rotten tuberculous lung-substance, which is likewise dependent in the form of shreds from its roof and walls.

This form of phthisis corresponds to the acute form of tuberculous intestinal ulceration, which runs its course without any reaction.

In other more common and less rapid cases, an inflammatory process, which must certainly be regarded as having a curative tendency, is established in the parenchyma around the softening tubercle or the primary tuberculous ulcer, and in the interstitial tissue amongst the secondary deposit. It gives rise to an albuminous grayish-white or somewhat reddish, tenacious and viscid product, which occasions the closure and finally the atrophy of the air-cells, and is identical with Laennec's *infiltration tuberculeuse gelatiniforme.* (See pp. 78, 79.) During this process the inner surface of the cavern becomes smoother and more uniform, and very often becomes covered with a thin grayish or grayish-yellow, thin, adhering investment of an apparently loose texture. This coating may sometimes, according to Andral, consist merely of the more solid portion of the pus contained in the cavity; in most instances this is, however, certainly not the case, but, as Laennec was the first to observe, there is a true exudation from the walls of the cavern like that which exposed or wounded animal tissues deposit on the surfaces of wounds or ulcers. This exudation is, doubtless, repeatedly thrown off, for if the process of tuberculous softening go on, neither it nor the adjacent wall of the cavern can meet with the conditions necessary for organization; it melts or becomes disintegrated, and mixes with the pus in the cavern, and another membrane is formed in its place, so long as the tuberculous process on the one hand, and the reactive inflammatory process on the other, continue in the tissue in a certain antagonistic degree and proportion. The caverns enlarge, in the manner we have already described, by the softening and breaking down of the secondary tuberculous deposit in the tissue of their walls, and by the confluence of several neighboring caverns into one. The parenchymatous bridges which traverse them are in a state of gelatinous infiltration, and contain tubercles, while externally they are coated by the above-named exudation.

In consequence of this process the lung-substance in the walls of the caverns becomes atrophied and converted into a more or less pigmentary, bluish-gray or blackish-blue, dense and tough layer of various thickness, the portion next to and lining the inner surface of the cavern being chiefly a whitish cellular tissue. To this the above-described exudation adheres, and through both these shine the bluish atrophied parenchyma and the vessels which are laid bare and obliterated by the cavern, and which appear as yellowish-white ramifying streaks; scattered crude or yellowish softened tubercles may also be observed. These tubercles gradually soften, and lead, on the one hand, to a gradual enlargement of the cavern, while, on the other hand, they impede any comprehensive process of consolidation, since they perforate the cellular investment of the cavity. The internal surface of the whole cavern is even and tolerably smooth, except at the spots where there are these new tuberculous excavations.

The pulmonary vomica in this condition is analogous to the tuberculous ulcer of the intestine with gelatino-lardaceous thickening of the submucous tissue on which it is situated.

The caverns naturally present the most manifold differences in reference to their size and number. The cases are not rare in which an abscess attains the size of a duck's egg, or of the fist, or even involves a whole lobe. When it is very large, the probability is that it has been formed by the confluence of several smaller caverns. The largest abscesses occur, with few exceptions, in the upper lobes, where, as we have already remarked, and especially in their upper third and at their apices, tuberculous deposits usually first occur, and where they first begin to soften.

It is a question of especial interest to ascertain how far the individual structures entering into the composition of the lung are involved in this destructive process, and above all, how the *bronchi* and *blood-vessels* within the tuberculous abscess are affected, what mode and form of destruction they undergo, and what destructive consequences follow when the cavern, in making its way outwards, finally reaches the *pulmonary pleura.*

The capillary bronchi undergo the same softening as the true lung-substance, for they, or at least their walls, are the seat of tuberculous deposition, and their mucous membrane becomes the seat of tuberculous infiltration (bronchial tuberculosis, p. 38) during the softening of the pulmonary tubercle, just as we observe in the larger bronchial tubes in the neighborhood of a tuberculous abscess. The capillary vessels become obliterated in the tubercle, and are exposed to the same softening process as the cellular strata surrounding them. If the cavern should now enlarge, the bronchial tubes become destroyed in the same proportion with the surrounding parenchyma, and it is only when the destruction of the tissue has attained a certain degree that an opening is effected into the tubes, and a communication established between the bronchi and the cavern. We only find bronchial tubes of a comparatively large size opening into the caverns, for the smaller ones are compressed by the tubercle deposited in their walls and in their immediate vicinity, or by the products of interstitial inflammation, or they are closed by catarrhal tumefaction of the mucous membrane, or by tuberculous infiltration. Their mouths remain freely open in places where compression cannot affect them, in consequence of greater and more resistent thickness of their walls, or of their having a larger calibre. The number of bronchial tubes opening into a cavern is generally proportional to its size. They constantly open with a round or an oval fissure-like mouth, according as they are more or less transversely or obliquely situated in relation to the walls of the cavern, or are only ulcerated on one side. When the bronchial opening is recent, it commonly presents an ulcerated appearance, but subsequently, when the cavern has acquired a dense callous wall, it is bounded by a puckered border of mucous membrane in a state of gelatinous infiltration, which is analogous to the serrated, puckered, and similarly infiltrated border of mucous membrane which surrounds the callous tuberculous ulcer of the intestine. The mouth of the bronchial tube opens in exactly the plane of the wall of the cavern, and never projects beyond it.

The blood-vessels present, as it were, the very reverse condition. The bronchial vessels are usually obliterated and thrust aside, and run along the walls of the cavern as ligamentous, projecting, yellowish-white, ramifying cords, and those of an arterial nature, even when in this condition, for a long time resist the destructive processes which are here in operation. A partially or entirely obliterated vessel, enveloped in atrophied lung-substance, is usually found in the bridges which run across the cavity of the abscess. It often, however, happens that, before the vessels are obliterated, they are laterally denuded of the surrounding tissue and of their cellular sheath; the two inner coats then soon give way, and occasion the pulmonary hemorrhages which, as is well known, occur in the course of phthisis.

There are two circumstances under which the caverns may reach the pulmonary pleura; either when they are originally formed in the peripheral portion of the lung, or when they were originally deep-seated, but have attained a considerable size in an outward direction. The first is very rarely dependent on the softening of tuberculous granulations, but, as we shall presently show, is much more frequently a consequence of the softening and breaking down of tuberculous infiltration. In either case the pulmonary pleura may finally be destroyed, and this may occur in different ways and with different consequences. If there are no adhesions at the point where the cavern reaches the pleura, this membrane, after being denuded on its pulmonary surface, will be converted into a yellowish-white eschar, which extends over a greater or smaller portion of the cavern, and either becomes torn or else loosened along its circumference and falls out in an entire piece; in this way tuberculous pus and atmospheric air find their way from the bronchial passages into the pleural cavity, and give rise to *pleurisy with pneumothorax*, and usually to speedy death. Mere cellular adhesions cannot prevent this termination; they are, in part, mechanically loosened by the effusion from the cavern, and being involved in the pleuritic process, they are, in part, likewise destroyed in the exudation. If, on the other hand, there are thick adhesions, that is to say if the lung is bound down by dense, thick, callous, cellulo-fibrous, and fibro-cartilaginous pseudo-membranes, such as occur especially about the upper lobes and their apices in consequence of previous pleurisies, then the pulmonary pleura which has coalesced and become identified with these false membranes may sometimes be laid bare to a considerable extent without perforation and the above-named consequences ensuing. But the tuberculous destruction is usually limited by these callous bands; cases occasionally occur in which even these are perforated; irritation is set up in them at one or more spots; they soften, become tuberculous, and suppurate, layer after layer; in this way they finally become perforated, and the tuberculous process attacks the wall of the thorax, insidiously advances to the ribs and soft parts, and at length reaches the outer surface of the chest, or even of the neck (Cruveilhier), in the form of a tuberculous sinus variously combined with caries of the ribs, the sternum, and the vertebræ.

Tuberculous Infiltration, when associated with the above-described metamorphosis of interstitial tubercle, usually softens with very great rapidity, and by hastening the progress of the disease, constitutes what is termed florid, or, by English writers, galloping consumption. It causes the most frightful destruction of the pulmonary tissue, and gives rise to caverns of irregular form, which are surrounded by rotten, and as it were corroded parenchyma, infiltrated with tubercle and breaking down into pus. Tuberculous infiltration is most commonly deposited in the superficial portions of the lungs, and hence it is the caverns arising from this variety which most frequently open into the cavity of the pleura. There are several ways in which this may take place.

(*a*.) The pulmonary pleura may be puffed up by the air rushing into the cavern, and may be violently peeled off the tuberculously-infiltrated parenchyma for some distance beyond the extent of the cavern, so as to form a flattish, round bulla, which finally bursts.

(*b*.) It may be converted, as we have already shown, into a yellowish-white eschar, which either tears or becomes detached unbroken.

(*c*.) Both the pleura and the infiltrated parenchyma surrounding the cavern may be attacked with gangrene, and become changed into a dirty-brownish, or greenish, pulpy, shreddy, fetid mass.

This last-named termination is especially worthy of notice, as it may occur not only near the surface, but also in the deep-seated portions of the tuberculous infiltration, especially around a pre-existing cavern. Moreover, in consequence of the frequency with which intense tuberculosis of the bronchial glands is combined with tuberculous infiltration, it may occasionally happen that a communication may thus be established between a deep-seated pulmonary cavern and a cavern in a bronchial gland.

The *contents* of tuberculous caverns present many differences. Sometimes, and especially when the infiltrated tubercles begin to soften, these caverns contain a yellow and somewhat thickish pus; more frequently, however, they contain a thin, whey-like fluid (tuberculous ichor), in which may be observed numerous grayish and yellowish, friable, cheesy, purulent flocculi and particles, whose quantity, however, is not in itself sufficient to explain the profuse expectoration which so often occurs in phthisis. This fluid is often of a grayish-red, or reddish-brown, or chocolate color, from the admixture of blood; or of an ash or blackish-gray color, from the pigment which it takes up during the softening of the tissue. Moreover, the caverns sometimes contain smaller or larger fragments of lung, resembling the parenchyma contained in their walls, and chalky concretions are occasionally found in them. In other cases they contain coagulated or fluid blood in various stages of discoloration.

This metamorphosis of pulmonary tubercle, in its twofold form, constitutes, as has been already observed, *tuberculous pulmonary phthisis*. If we now direct our inquiry to the state of the lung-substance around the tubercles and their abscesses, and from thence to the other organs and systems, in a distinct and uncomplicated case of this nature, we shall arrive at the following conclusions, in addition to what has been already stated, as the result of an anatomical examination when considered in reference to the living organism.

In the upper lobes, and especially in their upper third, there is usually a large cavern, surrounded inferiorly by several smaller ones, some of which communicate with it; between these are yellow tubercles in the act of softening, and gray tubercles just becoming opaque and discolored, whilst in the lower portions, as well as in the inferior lobes, there is a comparatively small sprinkling of gray, crude, tuberculous granulations.

The lung-substance between the tubercles is found in various states, according to the progress made by the disease. It may be normal, but generally there is a vicarious emphysema developed in its superficial portions, while the deep-seated parts are not unfrequently hyperæmic, or in a state of œdema. It is, however, sometimes atrophied, and this is a more important change, owing in part to interstitial inflammation, in part to the obliteration of the bronchial tubes and air-cells, in consequence of the pressure exerted on them by the accumulated tubercles, and in part to the occlusion of the bronchi by the blennorrhœal mucous secretion when bronchial catarrh is simultaneously present. Inflammation (croupous pneumonia), which sometimes attacks the greatest portion of the non-tuberculous parenchyma, is also an important change; it appears partly as a brownish-red, and partly as a grayish-red hepatization, which is everywhere converted into yellow tuberculous infiltration, which becomes dissolved, and collects in vomicæ; or the pneumonia may cause the deposition of a gelatino-glutinous product. (See p. 73.) In well-marked cases of this nature, the lung appears very bulky, and is coated with a grayish-yellow and generally thin pleuritic exudation, through which and the pulmonary pleura may be seen the peripheral tuberculous infiltrations, and the emphysematous patches amongst them.

In the *larynx* we find tuberculous ulcers, which vary in number and extent; and, associated with them, we find aphthous erosions, especially on the tracheal, and sometimes also on the pharyngeal mucous membrane.

The *bronchial tubes* proceeding to and from the caverns, exhibit streaks of mucous membrane in a condition of tuberculous infiltration, and are themselves filled with tuberculous matter; moreover, they are always in a state of catarrh, with reddening and softening of their mucous membrane, and with a muco-purulent secretion, which constitutes the greatest part of the sputa which are expectorated in the course of phthisis. The *bronchial glands* are enlarged, and more or less tuberculous.

Externally we find *pleurisies* which present great variety in their extent, and in the character, mode of organization, and consequences of the exudation. They are the causes of the very acute pains in the chest to which phthisical patients are subject. Unless when they arise from superficial pneumonia, they are generally developed during the softening of the tubercles and the formation of the caverns, and are associated with the inflammatory reaction that is established in the adjacent interstitial pulmonary tissue. The most constant seat of these pleurisies is the conical apex of the pleura and the surface of the upper lobes generally; they thus correspond to the starting-point of tubercle and of its metamorphosis. They deposit an exudation which becomes organized into fibro-cellular cords, or into a thick, compact, fibrous investment, which covers the upper lobes from the apices downwards, in the form of a hood, nd diminishes in thickness from above downwards, causing the lungs to dhere firmly to the costal walls, and thus affording a protection against he perforation that might otherwise be caused by large caverns.

With rare exceptions, we find *tuberculous intestinal phthisis* associated ith pulmonary phthisis, and although the former is usually only a econdary affection, dependent upon the pulmonary phthisis, it sometimes xceeds it in the rapidity of its progress, and rapidly occasions very great nd exhausting ravages. As a general rule, the lower portion of the leum is the part originally attacked, and from thence the ulcers extend pwards along this division of the intestine, and downwards over the olon; in their progress upwards the ulcers sometimes reach as far as the tomach. Frequently, however, and especially at certain times, the uberculosis not only predominates in the colon, but is almost exclusively onfined to it, the ileum entirely escaping; and sometimes we may readily erceive that the ulcers which are simultaneously present in the ileum re of a more recent date than those in the colon. In addition to the uberculous ulcers on the intestinal mucous membrane, we also find that he corresponding *mesenteric glands* are more or less tuberculous.

The mucous membrane of the alimentary tract, especially of the tomach and large intestines, is also in a state of more or less developed lennorrhœa; and, towards the end of phthisis, an *acute softening* of the nucous membrane of the great *cul de sac* of the stomach is not of unommon occurrence.

The *liver* is very frequently affected; the condition known as nutmeg iver, and depending on a morbid separation of the yellow and reddish-rown substances, with a preponderance, and more or less fatty degeneation of the former, is extremely common, and so, also, is the true fatty iver. These changes in this organ are not peculiar to phthisis,—that is o say, to the softening of the tubercles and the tuberculous ulceration f the pulmonary tissue,—but are associated with tuberculous disease enerally.

The *spleen* exhibits no constant change which stands in any essential onnection with tuberculous ulceration of the lungs.

The right side of the *heart* appears sometimes to be dilated, in conseuence of the impermeability of the lungs, induced by tubercle and its onsecutive diseases; it is, however, much more frequently remarkably mall, pale, and devoid of fat, in consequence of the anæmia which acompanies phthisis in its progress. In the former case we find stasis and ccumulation of blood in the right side of the heart, and from thence, in he whole venous system; in the latter, there is a general deficiency of lood, and a contracted aortic system.

The *central organs of the nervous system* exhibit no essential anomaly, lthough, as a consequence of acute phthisis, we not unfrequently observe yperæmia of the brain and its membranes, and recent serous effusions nto the ventricles, associated with white (hydrocephalic) softening of the erebral substance.

The *muscles* are all emaciated in an extreme degree; the fat is, in ost cases, almost entirely consumed, and the *cellular tissue*, especially n the extremities, is very often in an infiltrated condition.

Tuberculous pulmonary consumption is unquestionably *curable*, as we may infer from the appearances not unfrequently observed in the dead bodies of persons who formerly had more or less suspicious thoracic affections, and subsequently recovered. It is only by the investigation of the conditions under which these natural cures take place, that we can hope to arrive at a truly rational mode of treatment, and the results will be the more beneficial when directed against the tuberculosis generally, and not merely against the pulmonary abscesses. Pulmonary phthisis, or tuberculous ulceration of the lungs, can only be healed when the general disease, and consequently the local process on which the ulceration depends, is eradicated. There are incontrovertible facts to show that, under these conditions, pulmonary abscesses may actually heal in various ways.

a. The reactive inflammation of the interstitial tissue in the vicinity of the caverns gives rise, as has been already mentioned, to a gelatinous infiltration which causes an obliteration of the air-cells. By this means the whole of the adjacent parenchyma is converted into a dense, fibro-cellular layer of varying thickness. While this is taking place, the exudation, which is deposited by the same inflammatory process on the walls of the cavern, becomes organized from this fibro-cellular tissue into a smooth serous membrane. The whole cavern is now converted into a *cellulo-serous cavity*, whose inner surface secretes a serous, viscid fluid resembling synovia. The bronchial tubes, which open into these cavities, present a peculiar character, for the serous membrane lining the cavern, and the subjacent fibro-cellular tissue project beyond the outer stratum of the bronchial tubes at their openings, and their mucous coat hangs forward with a wrinkled, somewhat inverted free edge into the cavity.

More commonly, however, we find the caverns lined with a *villous, cellulo-vascular, more or less deep red layer resembling mucous membrane*, which is intimately connected with the subjacent tissues. It appears in a constant state of irritation; and, as we generally find in caverns with which large bronchial tubes communicate, its conversion into a smooth serous membrane appears to be impeded by the irritation induced by the constant entrance of atmospheric air. An already formed serous investment may doubtless be again reduced to this cellulo-vascular mucous membrane-like state, in consequence of this continuous influence. It secretes a torpid, muco-serous fluid, and it is not unfrequently observed to be covered with fresh exudations in consequence of higher degrees of irritation. It is extremely probable that these processes of irritation, associated with other causes presently to be described, effect the gradual diminution and finally the closure of the caverns. In cavities of this sort, the form of the bronchial openings is somewhat different from that which has been already described; for the bronchial mucous membrane coalesces with the lining texture of the cavern, which is analogous to its own tissue, and they merge into one another without any apparent line of demarcation.

Osseous laminæ are sometimes developed under the serous investment, in like manner as in the cellular tissue beneath normal serous membranes.

In these caverns an event not unfrequently takes place, which very often proves fatal on its first occurrence; this is the *hemorrhage* which is met with in caverns of this construction, and which always springs from the larger branches of the pulmonary artery traversing the walls; these branches often remain permeable, and become opened for a considerable extent on the side towards the cavity. There are two different conditions which may give rise to the opening of these arteries.

(*a.*) They either undergo an aneurismal dilatation in consequence of the absence of support in the direction towards the cavern, and finally tear at this point, without any further change in the texture of their membranes;

(*β.*) Or the delicate cellular sheath of the vessel participates in the irritation of the adjacent investment of the cavern; the process extends to the fibrous coat, which becomes relaxed and infiltrated with gelatinous matter; and the vessel finally gives way, a previous dilatation of its coats being sometimes but not always observed.

A circumstance deserving of notice sometimes accompanies these hemorrhages. The extravasated blood in the cavern coagulates into a fibrinous clot, which completely fills it, and is attached to a pedicle, which is seated in the rent in the vessel, and is continuous in both directions with the cylindrical clot in the artery. The cavern may certainly contract around this clot of fibrin when, in the course of time, it has become shrivelled and finally cretified; but as the cavity in its previously described state must be regarded as innocuous, and may be closed in another and a simpler manner, this method of cure, except in cases where hemorrhage takes place into a cavern which does not communicate with a bronchial tube, must always be regarded as dangerous, and only of actual use, insomuch as the fibrinous coagulum affords a support for the vessels in the walls, and prevents subsequent hemorrhages, which might occur before the cavity had closed by the ordinary way.

The above-described cavern must be regarded as a cured pulmonary abscess; but the cure may progress further, till there is perfect cicatrization.

b. This occurs in the following manner:—If the abscess be not too large, it closes by a gradual approximation of its walls, which finally come in contact and coalesce. We then find, in place of the previous cavern, a cellulo-fibrous stripe, in which the bronchi end in blind sacs. This is of most frequent occurrence in the apices of the lungs, where the coexistence of open caverns and the presence of obsolete and cretified tubercles indicate the nature of the processes that is here going on. The obliteration of a cavity of considerable size always occasions a corresponding depression of the surrounding parenchyma, and a cicatrix-like folding and puckering of the pulmonary pleura, which is most frequently and distinctly observed in the case of those cavities which are often superficially situated quite in the apices of the lungs. The thorax is also depressed to an extent corresponding with the size and number of the closing vomicæ, and is obvious from the flattening and slight depression so frequently observed in the clavicular region.

This process is undoubtedly favored very essentially by certain circumstances, amongst which we may enumerate the local depression of the thorax, the contraction of its cavity in consequence of the diaphragm being abnormally pressed upwards by the contents of the abdomen, the development of emphysema in the parenchyma surrounding the cavern, and bronchial dilatation. It has been proposed and attempted to produce these conditions artificially, by way of treatment, in various and sometimes violent ways: we have already discussed (in the first volume) the admissibility of these methods of treatment, their modes of action, and the consequences to which they may give rise.

When the healing process is rapid and continuous, the cicatrix sometimes encloses chalky concretions of various sizes, formed by the inspissation of tuberculous pus in the cavity.

(*c.*) The cavern, instead of cicatrizing in the above-described manner, may be filled up with a roundish or irregularly branched mass of fibro-cartilaginous structure, in which the bronchi terminate in blind sacs. This is effected by the conversion of its cellulo-fibrous walls into a fibro-cartilaginous callus, which continues to grow thicker. The cicatrix-like puckering of the surrounding parenchyma is generally in this case very inconsiderable.

This fibro-cartilaginous mass may sooner or later be converted into a very compact osseous concretion of corresponding form and size.

2. The second metamorphosis which pulmonary tubercles undergo under favorable conditions, is their *cretefaction.* After their softening has began or is perfected, they gradually diminish in volume and become converted into a yellowish-white, or grayish, or blackish-gray, smeary, chalky paste, and finally into a calcareous concretion. This concretion is situated, according to the intensity and extent of the process of reaction which is set up in the neighborhood of the softened tubercle, either in obliterated pulmonary tissue, or in fibro-cellular, or callous, fibro-cartilaginous capsule. Here also cicatrix-like puckerings of the parenchyma occur over the cretified tuberculous masses.

Tuberculous infiltration may also undoubtedly undergo this metamorphosis, for we not unfrequently meet with paste-like masses of chalk, together with cretified tuberculous granulations in the apices of the lungs, and corresponding in size and form to a pulmonary lobule; they are surrounded by a very delicate sero-cellular capsule, formed of condensed interlobular cellular tissue, and most probably are cretified *lobular*, tuberculous infiltrations.

3. Finally, pulmonary tubercle, when *in the form of crude gray granulations, may become obsolete*, shrivelled up, and abortive. It is then changed into opaque, bluish-gray nodules, having the resistent power of cartilage, which are incapable of any further metamorphosis. This destruction of the tubercle is either general, or it is combined with the process of cretefaction, the central portion or nucleus being converted into a chalky concretion encysted in the obsolete peripheral layer of tuberculous matter.

From what has been already stated, it follows that pulmonary tuberculosis may be cured by phthisis with the elimination of the tubercle; but the two last-described metamorphoses, opposite as they are to one another, constitute more direct healing processes. Any one of them may take place under favorable conditions, and as a general rule, they are all found in one and the same individual, for we find associated together cellulo-fibrous caverns, their cicatrices, and cretified and obsolete tubercle. They are generally all found imbedded together in obsolete parenchyma, infiltrated with black pigment.

Tuberculosis is either an *acute* or a *chronic* disease. In *acute* cases it attacks both lungs simultaneously, and frequently other parenchymatous organs and membranes, giving rise to peculiar symptoms resembling those of typhus; the tubercle is the product of tuberculous dyscrasia of the blood developed in a very high degree. The tuberculous mass is, in some cases, deposited at once, and in others at different intervals, which rapidly succeed one another, and are indicated by paroxysmal exacerbations: it is formed of gray, crude granulations, which are either very minute, vesicular, and transparent, or in some cases, as large as millet-seeds. The tubercles are always very numerous, discrete, and uniformly scattered through the lung-substance; it is only rarely that we find them accumulated and confluent at individual spots, and in these cases they are all in the same stage, namely, that of crudity. Moreover the lung is in a state of hyperæmia, œdema, and emphysematous textural relaxation; the hyperæmia occasionally passes into pneumonia and hepatization.

In most cases it only attacks the lungs after tuberculous disease has advanced in them to the stages of softening and ulceration (vomica), and after it has existed for a longer or shorter period in its favorite locality,—the apices of the lungs—in the state of more or less circumscribed, insidious tuberculosis. A pre-existing chronic tuberculosis of the lungs is generally the predisposing cause of the acute production of tubercles in those organs. It proves fatal in consequence of the hyperæmia and of the subsequent œdema to which it gives rise, in consequence of the violent production of emphysema, or from paralysis of the lungs.

Chronic tuberculosis either deposits its product imperceptibly, or else as crises of a mild general disease, with symptoms of moderate vascular excitement, and recurring at intervals. In accordance with this view we find tubercles of various ages and stages; and at the extreme points of the diseased lung-substance we have the two extreme stages of tubercle; at the apices, where the tubercles are first developed, we have caverns; and at the lowest portion we have recent, crude, tuberculous granulations; between these we have dissolved tubercles next to the caverns, and lower down such as are just beginning to soften.

It either proves fatal in the form of phthisis through exhaustion and tabes, or through some of those accidents which we have already described as liable to occur in the course of phthisis, as for instance the supervention of pneumonia with a tendency to tuberculous infiltration (hepatization), hyperæmia or œdema of the lungs, hyperæmia of the brain and serous effusion into its ventricles (hydrocephalus, serous apoplexy), tuberculous meningitis, exudative processes in the neighboring mucous canals, as the trachea or œsophagus, purulent metastases, or the supervention of acute pulmonary or general tuberculosis.

The tuberculous habitus in general, and more especially the irritable scrofulous habitus, are the stamp indicating a predisposition to pulmonary tuberculosis; the torpid scrofulous habitus more commonly gives rise to bronchial tuberculosis. The well-known (phthisical) conformation of the chest which predisposes to pulmonary tubercles, is by no means invariably present; its peculiar relation to tuberculosis is unknown, and any connection between the smallness of the respiratory organs in a contracted thorax, and the development of pulmonary consumption, is only hypothetical. Tubercles are often developed in the lungs of individuals, independently of any marked external influences, and then form *constitutional pulmonary tuberculosis* and *pulmonary phthisis.* On the other hand, they may arise independently of this constitutional dyscrasia, in consequence of appreciable noxious influences, which induce either a purely tuberculous condition of the juices, or a modification, that is to say, a combination of this state with some other. This is *acquired* tuberculosis, which is either *pure*, or more or less *modified and combined*, such as follows the exanthemata and impetigo, gonorrhœa, syphilis, and anomalous gout, and occurs in drunkards, after the suppression of normal or habitual discharges, as, for instance, of the menses, after the cure of inveterate ulcers, &c.

The inveterate forms of dyscrasia deposit different varieties of tubercle, which have not hitherto been fully described; they occasionally terminate in *hemorrhagic tubercle.* The tuberculosis is distinguished from the ordinary forms by commencing at an uncommon part, by its unequally attacking the most different parts of the lungs, by the deposit being accumulated in circumscribed or grape-like branches, by its very considerable amount, and by its peculiar, dirty-gray or leaden color with a greenish sparkling appearance. (See Vol. I.)

Cancer of the lungs is sometimes deposited in a form resembling tubercle; we must carefully avoid confounding these morbid growths.

Pulmonary tuberculosis, like tuberculosis in general, is excluded by all the conditions enumerated in the first volume, especially by diseases of the lungs, attended with atrophy, emphysema, bronchial dilatation, excessive condensation, compression, obsolescence or obliteration of the tissue.

6. *Cancer of the Lungs.*—Cancer occurs in the lungs both in the form of *carcinoma medullare* and *carcinoma fasciculatum, seu hyalinum.* The latter is extremely rare, but the former is comparatively common, and it is to it that the following observations apply.

a. It most commonly occurs in the form of *roundish, separate masses,* varying from the size of a hemp-seed to that of the fist, and occasionally being even larger, and enclosed in a very delicate cellular capsule; they are composed of gelatino-lardaceous or lardaceo-encephaloid or true encephaloid parenchyma, and hence they vary considerably in consistence; they are usually white, but are sometimes of a grayish-red, or dirty yellowish-gray color. They are generally scattered in very considerable numbers throughout the lungs, both near the surface and deep in the texture, and when they are contiguous to the pulmonary pleura, they undergo a flattening or depression. The injury of the surrounding parenchyma is limited to its being displaced and compressed in the immediate vicinity of the adventitious product. It is only very seldom that it undergoes ichorous disorganization, in which case the accumulated cancerous ichor makes its escape by communicating with the bronchi.

It usually proves fatal by the exhaustion induced by its excessive growth, and by the high degree of general cancerous cachexia from which the growth originates. Pulmonary œdema and hydrothorax commonly supervene, either with or without simultaneous cancer of the pleura.

It very rarely occurs in the lungs as primary cancer, that is to say, as the first in a series of successive local cancers; it almost always exists in association with other, and generally many cancerous deposits of older date, distributed over several organs; and is often developed with great rapidity after the extirpation of large cancers. It is chiefly combined with cancer of the pleura, with which it is usually simultaneously developed, or with cancer of the mediastinum, or of the mammary gland, the liver, the kidneys, or the osseous system.

b. The occurrence of pulmonary cancer as a *special form of tubercle* is very rare, and is never met with unless when there is cancer in some other organ. It presents itself in the form of tubercles or nodules of the size of a millet or hemp-seed, which, as far as we yet know, may be distinguished from other tubercles by their bluish white color, their softer consistence, their aggregation in groups, and a difference in their elementary structure and composition. They sometimes exist in association with a *retrograde genuine pulmonary tuberculosis.*

c. Cancerous matter is very rarely *infiltrated or effused into the air-cells.* When it occurs in this form it is the product of a pneumonic process, which, under the influence of a dyscrasis excited by the extirpation of cancer, assumes the external characters, and the elementary structure of carcinoma; the lung in this case appears hepatized with cancerous matter.

Medullary cancer of the lungs is sometimes more or less blackened by a pigment which enters into its composition; the medullary nodules are marked with brown, blackish-blue, violet, or black spots or stripes, or are completely and thoroughly black, constituting melanotic cancer—cancer melanodes—of the lungs. We have never met with it except in association with general and, in fact, with very acute medullary cancer.

SUPPLEMENT.

1. *Diseases of the Thyroid Gland.*—As a general rule, the thyroid gland is liable to few diseases, and of these diseases we are almost as ignorant as we are regarding the structure and the function of this organ.

It very frequently presents anomalies of *size*, being often very much enlarged. The *augmentation of size* is sometimes transitory and rapid, as, for instance, when it depends upon congestion or inflammation, and sometimes in the case of lymphatic goitre; or there may be a persistent gradual increase, as is observed in the more advanced stages of goitre. It either attacks the whole gland uniformly, which then retains its original shape, or one lobe only, or a small part of one may be the only portion affected, so that the pressure which the gland naturally exerts on the trachea and larynx is variously increased in extent, and may affect not only the pharynx and œsophagus, but also the great vascular and nervous trunks on both sides of the neck, and even the trachea and bronchi, and the blood-vessel within the thorax. Those forms of enlargement are rarer, but at the same time more important, in which the thyroid gland tends to surround the œsophagus like a ring, and in which the isthmus grows downwards so as to form a middle lobe, which descends along the trachea behind the manubrium sterni into the thoracic cavity; in the latter case it becomes transversely contracted when opposite the semilunar notch, but expands immediately below it (*asthma thyroideum*).

The *diminution of size* or *atrophy* of the thyroid gland is an affection of little interest.

Hyperœmia of the thyroid gland is not unfrequently observed, and most commonly occurs when there is some mechanical impediment to the emptying of the vena cava descendens and of the right side of the heart. Under these circumstances it may be either transitory or persistent. It may be recognized by the dark color of the gland, its abundance of blood, its looseness of texture, and its swollen condition (hyperæmia, congestive turgescence). *Apoplexy* of this gland, when its texture is normal, is extremely rare.

Inflammation of the thyroid gland, as a *primary* affection, is of very rare occurrence, at least as an object of anatomical observation. But we sometimes find what are termed metastatic *abscesses* in it, especially when there are numerous similar deposits in other organs, consequent on puerperal uterine phlebitis. Abscesses of the thyroid gland may give rise to a deposition of pus in the mediastina, or they may open into the trachea, or, which is most commonly the case, they may enter into the œsophagus on its left side.

The most common disease of the thyroid body is that to which we apply the word *struma* (using the term in its strict signification), and its most striking characteristic is, as we have already mentioned, an augmentation of size. In the slighter degrees in which it usually occurs, it presents a very simple change of texture depending on a more decided development of the cellular structure of the organ. This occurs either equally through the whole gland, which then everywhere contains cells of equal size, or else we observe one, several, or very many isolated or agglomerated cells larger than the others, which are converted into roundish elongated cysts, with delicate membranous walls, and contain a gummy or glue-like, yellow, brownish or greenish matter (colloid). If this matter has attained a certain consistence, the cut surface of the gland presents a lardaceous appearance, and communicates a peculiar waxy and doughy feeling; the organ is at the same time pale and anæmic, and presents a marked increase of size without any disproportion of form.

There are certain unknown conditions under which, on the one hand, the secretion contained in the dilated cells undergoes modification either from the beginning or during the progress of the disease, or, on the other, the walls undergo a striking change. In the former case we find gelatinous or albuminous substances, of a whitish, gray, or flesh-red color, deposited in the form of concretions, whose coat may be peeled off, or they fill the interstices of an extremely delicate cellular network of new formation. In the latter case the walls of the cells increase in thickness, and the cells become developed (hypertrophied) into sero-fibrous cysts, which may contain various matters besides those already named, and which often attain an astonishing size. These changes constitute those forms of struma, which are known as *struma lymphatica* and *struma cystica*.

There can be hardly any doubt that these processes are essentially based on irritation, for repeated inflammations attack the walls of the dilated cells, and especially of the above-named cysts, during the ordinary progress of the disease, although they doubtless often pass unnoticed. Here, as on normal serous, and fibro-serous membranes, they deposit the most varied exudations, and in consequence of the newness of the tissues, these are often hemorrhagic, and accompanied by the separation of large clots of fibrin. These, together with the walls of the cyst, undergo all the same metamorphoses as occur in the exudations and the walls of normal serous sacs (see Vol. III.), even to chalky transformation and ossification. The cysts in this manner not unfrequently become perfectly obliterated by contracting around the exudation, and we then find tough, somewhat voluminous, nodular, osseo-cartilaginous, chalky concretions imbedded in the gland.

True effusion of blood not unfrequently takes place into the cavities of the dilated cells and of the cyst.

The tendency to *cyst formation*, exhibited by the parenchyma of the thyroid gland, extends in a remarkable manner to the adjacent cellular tissue, for in no situation do we so frequently meet with small or large cysts with serous, gelatinous, or glue-like contents, as in the neighborhood of this organ.

All other adventitious growths, excepting the above-named serous, fibrous, cartilaginous, and bony productions, are extremely rare in the thyroid gland; thus *tubercles* are scarcely ever found in it, and *medullary cancer* only very rarely.

2. *Diseases of the Thymus Gland.*—Anomalies of the thymus gland are even rarer than those of the thyroid body; the only abnormal conditions with which we are at present acquainted are a more or less considerable increase of its size in new-born children, and its persistence to the fifth, sixth, or seventh year, or even to or beyond the age of puberty. Its abnormal enlargement is almost entirely restricted to children in whom we simultaneously observe a great predominance of the whole lymphatic glandular system, rachitis, and hypertrophy of the brain. It presents either two lateral, flattish, round, thick lobes, which descend on each side into the mediastinum posticum, or it forms a tongue-shaped mass which extends downwards on the pericardium, and rests on the right auricle. Whether the *thymic asthma* which has been recently described, and which occurs in delicate children, is actually dependent on the pressure of an enlarged thymus on the air-passages, or whether there is any essential connection between that disease and the thymus, are questions requiring additional observations and careful examination.

PART II.

DISEASES OF THE ORGANS OF CIRCULATION.

PART II.

ABNORMAL CONDITIONS OF THE ORGANS OF CIRCULATION.

WE may divide the above into Diseases of the Heart, including those of the Pericardium, and Diseases of the Arteries, the Veins, and the Lymphatics. Under the last head are included Diseases of the Lymphatic Glands.

I.—ABNORMAL CONDITIONS OF THE PERICARDIUM.

§ 1. *Deficiency and Excess of Formation.*

The *first-named* species of malformation manifests itself as a deficiency of the pericardium, occurring generally when the heart lies outside the thorax, although it is also met with when this anomaly is not present; but is then of less frequent occurrence. This deficiency is in almost every instance merely partial, consisting in the congenital anomalous position of the heart outside the thorax in a fissure of the pericardium, although it is not uncommon in some cases to meet with less marked traces of the same condition in the region of the larger arterial trunks and along the right layer of the mediastinum. The heart and the left lung lie, as a general rule, in one common large serous sac, which gives rise, at the place from whence the arterial trunks emanate, to the above-mentioned rudiments or traces, in the form of fatty mesentery-like folds.

The apparent deficiency of structure, induced by the firm adhesion of the pericardium to the heart, seems to have been mistaken by some older observers, for true deficiency of structure.

An Excess of Formation occurs in double monsters, where the pericardium is found to contain a double heart.

§ 2. *Deviations in Size and Form.*

The size and form of the pericardium depend originally upon the size and form of the heart; and likewise, although in a less degree, upon the calibre, number, and arrangement of the vascular trunks springing from the heart.

An *acquired dilatation* of the pericardium frequently occurs in consequence of an increase of the heart's volume, or of dilatation of the vascular trunks, especially of the aorta, and very commonly from morbid effusions, as for instance, the formation of inflammatory products in its cavity. In these cases the dilatation of the pericardium is *uniform*, and frequently, as in exudations, and more especially in enlargement of the heart, it is so considerable that the pericardium may extend in a diagonal direction from the anterior extremity of the second or first rib on the right side to that of the eighth rib on the left side, having its anterior surface pressed againt the sternum and the costal cartilages, reaching on both sides to the lateral walls of the thorax, and compressing the lungs, especially their lower lobes, in the posterior part of the thoracic cavity.

Moreover, in some few cases, a *partial dilatation* of the pericardium also occurs as a *Diverticulum* or *Hernia Pericardii.* This is in fact a hernial dilatation, occasioned by the penetration of the serous surface of the pericardium through different non-resisting or ill-protected parts of the fibrous layer, or through apertures in this layer, expanding into an appendage, which is attached to the pericardium by a pedicle or neck, and communicates with it by means of a narrow opening or canal.

Our museum contains two very instructive cases of the development of *Hernia Pericardii;* in these the diverticula are seated on the lateral parts of the pericardium. Hart's case affords an important example of the attachment of a large appendage to the anterior portion of the pericardium.

§ 3. *Interruptions of Continuity.*

To these appertain injuries inflicted by various penetrating instruments, and by the impaction of fragments of the sternum or the ribs; lacerations from severe concussions or contusions of the body; ulcers perforating from without inwards, &c.

§ 4. *Diseases of Texture.*

a. Inflammation is the most common form of disease of the pericardium, and is of the greatest importance, not only in itself, but also from the subsequent results to which it may give rise.

Inflammation may either be primary or secondary, being in the latter case metastatic or derived from the inflammation of neighboring structures. It may be either general or partial. It may be acute in its course, or, and such is more frequently the case, of a chronic form. The greatest variety is found to exist both in the quantity and the character of the exudation. The following remarks on the inflammation of the pericardium will be found to be in accordance with what has been already stated regarding inflammation of serous membranes in general.

1. From the importance of the subject, we will, in the first place, consider primary inflammation of the pericardium; next, its general and partial forms, the character of the peripheral coagula of the exudation, with the mode of their organization *generally;* acute and chronic inflammation with purulent exudation; chronic inflammation recurring in the false membranes; inflammation with hemorrhagic effusion; and lastly, and *specially*, inflammation with an exudation of a tuberculous nature.

Primary general inflammation of the pericardium, in accordance with what has been already stated, affects the parietal as well as the visceral surface, that is to say, the external serous investment of the heart and vascular trunks; it is more developed on the former than the latter, excepting in a few cases, and its peripheral coagula are more copious and abundant. Among the anatomical indications of inflammation most worthy of notice, we may especially mention injection, because it may here often be distinctly observed and investigated. The serous surface of the pericardium assumes in that case the appearance of soft, red velvet, having obliquely erect piles, and looking pale and turbid, as if infiltrated.

Partial Inflammation may affect any part of the pericardium, as will be seen when we have occasion to refer to the circumscribed organized inflammatory products,—the so-called milk-spots.

The peripheral coagula appear very fully developed and distinct upon the pericardium, in the forms that have already been generally described. Their free surface commonly appears as if covered with villous threads, which are either soft and lax, or stiff, and vary in character. Laennec has compared them to the inequalities remaining on two plates, which, after having been covered with a layer of butter, and laid against one another, have been quickly separated, and it is probable that this appearance gave rise to the terms made use of by the ancients, when they described the heart as *cor villosum*, *tomentosum*, *hirsutum*, *hispidum*, &c. Sometimes these shaggy masses are more or less accumulated at different spots, or ranged side by side, which is doubtlessly owing to the direction of the undulations produced in the serous effusion by the heart's motion. In many cases they may be aptly compared to the appearance presented by the dorsal surface of a bullock's tongue, whilst in others, the coagulum exhibits an areolar free surface, similar to that of the mucous membrane of the gall-bladder.

When the coagulable matter occurs *in larger quantities* in the serous portion of the effusion, it is found in some few cases in the form of roundish and somewhat flattened free bodies, about the size of a bean or hazelnut, and generally constituting a network between the heart and pericardium, to both of which it adheres.

The plastic coagula become converted into a cellular or cellulo-fibrous dense tissue, with a permanent thickening of the pericardium, corresponding to the intensity of the process; and the different loose filamentous adhesions, or close fusions of the heart and pericardium, which are so frequently observed, either partially or totally (according to the extension of the process) are formed by this tissue. Amongst the partial adhesions, we may specially draw attention to those of a circumscribed nature occurring at the apex of the heart, those occurring at different parts along the sulcus transversalis, and the adhesions of the pericardium in the vicinity of the arterial trunks. In the first of these spots, the connecting medium is often drawn into long threads or strings by the movement of the heart's apex, and the adhesion is thus at length broken through, in consequence of which we usually find an accumulation of long, shaggy, cellular tissue at that part, and on the opposite portion of the pericardium; the second class of adhesions derive importance from their ordinary combination with diseases of the valves, especially towards their margin of insertion; and the last from the evidence which their common occurrence affords of the frequency of pericarditis, which may prove of serious moment at the origin of the large vessels, as we shall subsequently have occasion to consider.

The *milk spots*, or *maculæ albidæ*, are appearances of frequent occurrence on the heart. They are occasionally met with on the inner surface of the pericardium, but most frequently on the serous investment of the heart. There can scarcely be a question but that they are products of a partial or circumscribed process of inflammation. They are pale, bluish-white, tendinous-looking spots or *plaques*, appearing, when closely investigated, to be glued or soldered to the subjacent tissue; on being torn or detached, the pericardium is brought into view, and is almost normal in its character,—not perfectly smooth, but having a dense and sometimes even an opaque tissue. They must be distinguished from many other diffuse opacities of common occurrence on the pericardium, which consist in an inconsiderable excess of structure,—hypertrophy, a slight thickening and condensation of the serous investment of the heart. They further manifest many different characteristics with respect to their size and distribution, the number in which they occur, their form and limitation, the surface and mode of attachment; finally, they occur more frequently on some portions of the heart's surface than on others.

The *size* of these spots varies from that of a silver groschen or a narrow stripe, or that of a silver thaler or more, so that one spot often spreads over a great portion of the heart's surface. A number of these spots are often found together, and they then blend into one another. Their *form* varies very much, and is extremely irregular; they commonly occur as narrow stripes along the coronary vessels in the sulcus longitudinalis. They usually expand into linear projections at their periphery, and are either sharply defined, or gradually attenuated into a very delicate membrane, as may be seen in moist preparations. Their *surface* is either smooth, even, serous and shining, or wrinkled, folded, pale, felt-like, and shaggy; the whole presenting a layer of newly-formed cellular tissue.

These milk-spots occur on every portion of the heart, but they are certainly more frequent on the right than on the left ventricle; they usually appear on the auricles in the form of stripes, and finally are met with at the origins of the arterial trunks, and more especially at that of the aorta.

In connection with the subject of milk-spots, we may notice the metamorphosis of a partial exudation into *fibroid granulations*, occurring about the size of a millet-seed. These are especially to be met with on the auricles, and on the corresponding portion of the parietal surface of the pericardium. Granulations of this nature are frequently situated on these spots.

Inflammations with purulent exudation are distinguished by the quantity of the effusion, and are important on many accounts, which will be subsequently considered.

Scattered accumulations of pus are of very frequent occurrence in the subserous layers of the pericardium.

It is only in very rare cases that the purulent exudation leads to suppuration of the pericardium.

Our museum presents an instance of an originally sero-purulent exudation, which appears to have gradually undergone the following remarkable metamorphosis. At the circumference of the left side of the heart, the pericardium is closely adherent, whilst a whitish, very turbid fluid resembling milk of lime is accumulated around the right side of the heart. The inner surface of the pericardium, and more particularly the outer investment of the heart, appear partially encrusted as with a sandy mortar, and partly covered with a white, smooth, gypsum-like coating.

Among the *chronic inflammations* those attacking the pseudo-membranes are especially frequent and important; here we find peripheral coagula of very considerable thickness, density and power of resistance, and of a fibroid texture; the pericardium itself acquires a considerable degree of thickness, and in cases where there is a resorption of the fluid, the two lamellæ of the peripheral coagula adhere together, and the heart becomes enclosed in a thick, tough, unyielding casing. *Hemorrhage* very generally accompanies the secondary exudations in this process.

Finally, when the necessary conditions are present, the exudation may be *tuberculous;* but this subject will be noticed when we proceed to the consideration of tuberculosis of the pericardium.

In every form of pericarditis the pericardium may become very much distended, in consequence of the great quantity of the exudation, and especially of its serous portion.

Osseous concretions are not unfrequently developed in the dense fibroid exudations occasioned by the process of chronic inflammation recurring in the pseudo-membranes. We shall have occasion to return to this subject in a future page.

2. In reference to the *secondary effects* produced in the organism by general pericarditis, among which we must especially place extensive inflammations of the large serous sacs, it is worthy of notice that several, as for instance cachexia and dropsy, usually occur at an early stage and in a high degree of development. These conditions are occasioned by the injurious influence exerted by the pericarditic process on the heart, in consequence of which the muscular substance of that organ is paralyzed, its color changed to a dirty brown or yellow, and a flabby condition induced, which admits of the texture being easily torn, and which speedily leads to (passive) dilatation of the heart. These phenomena are collectively the more striking in proportion as the pericarditis is chronic, and the exudation is purulent, hæmorrhagic, or tuberculous; the dilatation becomes more permanent, the more completely the coagula have been metamorphosed into a thick dense resisting tissue surrounding the heart.

Pericarditis, more especially when of a chronic form, is important in reference to the origins of the large vessels. It would seem, according to our view, that this disease, as far as it affects the cellular sheath of the vessels in the sub-serous cellular tissue, must induce paralysis of the elastic coat, dilatation of the aorta, and that form of spontaneous laceration of the vessels within the pericardium, which is so often found to occur.

3. Pericarditis frequently occurs in original combination with inflammation of other serous sacs, as for instance with pleuritis, inflammation of the synovial membranes of the large joints (Bouillaud), and very fre-

quently with pneumonia. It is, moreover, in like manner associated with endocarditis, and occasionally with carditis: during the later stages it is often accompanied with the first-named inflammatory processes, and also with meningitis.

Pericarditis is often a secondary affection associated in various degrees of intensity with other processes of exudation, and very frequently a slight reddening, injection, and an inconsiderable degree of effusion, are found to attack the pericardium at periods of complete exhaustion, and in consequence of extensive exudations. Acute pleurisies extend from the mediastinum to the pericardium, and centres of inflammation in the muscular tissue of the heart sometimes occasion general and sometimes partial inflammation of the pericardium.

Pericarditis, contrary to the results of the investigations of many observers, is frequently met with beyond the middle periods of life and even in advanced age.

b. Secondary Formations.

1. *Adiposity of the Pericardium.*—We not unfrequently observe an excessive accumulation of fat on the pericardium. This occurs in general not only in conjunction with an excess of fat in the heart itself, but also together with fatty accumulations in the abdomen, that is to say in the great omentum and its appendages, in the mesentery, under the costal pleura, &c., constituting a general condition of corpulence.

2. *Fibroid Tissue* occurs in the milk-spots and in the fibroid granulations, assuming the form of a thick exudation, having the property of resistance, and being of a very dense texture.

3. *Anomalous Osseous Substance* is scarcely ever developed, excepting in the above-named fibroid exudation, after the lamellæ have become fused together, and the pericardium has thus been made to adhere to the heart by this dense and resisting fibroid medium. This adhesive stratum is now covered by a deposit of tuberous uneven laminæ and bands, or thick, roundish, nodular masses. The space occupied by the deposits varies considerably, but the first-named of these forms of deposition occasionally extends so far as to cover the greater portion of one ventricle. The projections occupying the side next the heart frequently extend to the texture of the heart itself, displacing the muscular bundles, and appearing as if developed within them. The thick, round, nodular masses are generally observed in the neighborhood of the sulcus transversalis on the left side of the heart, being connected with an osseous concretion which usually has its seat at the margin of insertion of the mitral valve. They are consequences of a former state of endocarditis combined with pericarditis.

4. *Tuberculosis of the Pericardium.*—Tuberculosis rarely manifests itself in the pericardium in any other form than as a product of inflammation. Pericarditis gives rise to an exudation, whose peripheral coagula, after passing, wholly or in part, through various metamorphoses, merge into tubercle. It frequently happens in chronic inflammations of the exudation-deposits that the deeper or older strata have become tuberculous, while the more recent coagulum, which is becoming tuberculous, is covered by a secondary, villous, and shaggy deposit from the fluid effusion.

This form of tuberculosis of the pericardium, in accordance with what has been already stated regarding tuberculosis of the serous membranes generally, is not of a primary character, being usually associated with and dependent upon an earlier tuberculous condition, which has formed as it were the focus or starting-point of the disease, and has been manifested as tuberculosis of the lungs and bronchial glands, or as a chronic tuberculosis of some of the great serous membranes, especially of the peritoneum.

In this form of pericarditis there is always much serous effusion, which is undoubtedly increased by the inflammation being paroxysmally developed in the tuberculizing coagulum. This effusion frequently becomes hæmorrhagic, in consequence of such secondary exudations.

The tubercles, which are often of considerable size, and fused together into one aggregate mass, are occasionally seated close to the muscular tissue, into whose fibres they occasionally penetrate so far as to lead to much doubt regarding their original position.

This form of tubercle very rarely passes into the metamorphosis of complete softening, since death, when it ensues, is generally occasioned by the pericarditis, or the subsequent tuberculous secretions, or even by general cachexia. Occasionally one or more tubercles, or tuberculous masses, may certainly be observed to become disintegrated, but the process is seldom sufficiently prolonged to produce an abscess, or a corrosion,—tuberculous suppuration of the pseudo-membrane and of the pericardium itself. As we have already observed, the tuberculous exudation soon manifests its influence on the tissue of the heart, which, however rapidly the disease may prove fatal, is always found to be strikingly discolored, having generally acquired a dirty-brown color, and is moreover flabby and easily torn.

5. *Cancer* only affects the pericardium in a secondary manner; and, in most cases, only where secondary cancerous formations have been developed in the mediastinum. This secondary mass either spreads itself in the form of an infiltration of the fibrous layer of the pericardium over a large portion of its surface, or presses upon and into the tissue itself, where it becomes developed into roundish or flattened, teat-like nodules.

In the very rare cases in which cancer occurs in the pericardium, independently of the above conditions, it presents itself in the form of numerous, flattened, and roundish nodules. It then always occurs in combination with cancer of other serous membranes, especially of the contiguous pleuræ, and depends upon an excessive dyscrasia, developed by previous cancerous degeneration of different parenchymatous structures, and frequently exasperated by the eradication of large carcinomatous masses.

We have never met with any other form of cancer of the pericardium but the medullary.

§ 5. *Anomalies of the Contents of the Pericardium.*

Besides the anomalies already treated of, it remains for us to notice, among those which exhibit special points of interest:

Blood in a fluid or coagulated condition. It is almost always an arterial extravasation, and has been deposited by the spontaneous rupture of the left ventricle, or by a laceration of the origin of the aorta, occasionally also as the termination of an aneurism. The quantity extravasated seldom amounts, under these circumstances, to more than from 2 to 2½ lbs.

Serum is frequently accumulated in greater excess than the normal quantity, which varies from ½ oz. to 1 oz.; and it then constitutes *Hydrops Pericardii.* This accumulation becomes serious in proportion to its amount, and possesses greater importance where the other coexisting anomalies, by which dropsy is influenced, are inconsiderable. It is generally combined with dropsy of other serous sacs, anasarca, and œdema of the lungs, and usually has a common origin with them. Occasionally it predominates over these affections, and is especially the case in pulmonary phthisis. The period of its duration may be estimated by the extent to which the fat has disappeared from the heart, and its place been occupied by a serous infiltration of the cellular tissue, and in proportion to the extent of turbidity and swelling from imbibition, observed in the pericardium, and especially its outer surface, and to the decoloration and paleness of the substance of the heart.

The quantity of the *Liquor Pericardii* is often strikingly small, amounting to no more than what is barely sufficient to moisten the pericardium and the heart itself. The pericardium may even in some parts appear perfectly dry, of a yellowish color, and resembling parchment. We have remarked this appearance, which is devoid of importance, on the lateral portions of the pericardium, principally on its left side, where it had been brought in contact with the anterior parts of a lung in which emphysema has been developed.

We have never met with an accumulation of *Air* in the pericardium, *Pneumatosis Pericardii.* Most of the cases recorded, like the pneumatosis of other serous sacs, leave room for many doubts regarding their existence during life.

Free Bodies are of very rare occurrence in the pericardium. In a very remarkable case of pericarditis we discovered, in the serous effusion, numerous fibrinous, soft, yellow concretions of the size of beans or almonds, and similar to the latter in shape, which would no doubt have eventually been converted into elastic, tough bodies of fibroid tissue.

II.—ANOMALIES AND DISEASES OF THE HEART.

We will now proceed to consider the Anomalies and Diseases of the Heart, including those of the Valves; but wherever it may prove of great practical interest to acquire a more correct knowledge, both generally and specially, of the anomalies of the valves treated of in the different sections, we purpose, at the close of each, entering more fully into the details of the subject.

We will, moreover, consider simultaneously all original malformations of the heart and of the vascular trunks, not only on account of the unnatural connection existing between them, but also with a view of furnishing the premises necessary for the better comprehension of the appendix on cyanosis, which is subjoined to our remarks on the anomalies of the heart. In order, as far as possible, to facilitate a reference to the most important original malformations, we have arranged the following sections somewhat differently from those by which they are preceded.

§ 1. *Deficiency and Excess of Formation.*

Absence of the Heart—Acardia—is generally of very rare occurrence, but is a common phenomenon in Acephalia (absence of the brain), especially where there is an absence of the upper half of the trunk. It has only been observed in very rare cases where the nervous system is perfect and complete.

In the consideration of deficiency of the heart we include a series of deficient formations (arrested developments) which may be arranged as follows.

a. The lowest type of formation is that in which a single cavity without valves represents a ventricle in which a dilatation of the vena cava appears as the rudiment of an auricle. The latter is membranous, and the former has only thin muscular walls and weak trabeculæ.

b. Next we have a heart consisting of one ventricle and one auricle, with simple vascular trunks, into the former of which opens an aorta, and into the latter a vena cava. In many cases this formation approximates to the succeeding one in which there are two auricles with a single ventricle.

c. In this form there is a single ventricle and one auricle, which is either partially or wholly divided into two cavities by means of a partition wall. The arterial and venous trunks may be either single or separated.

d. Here a capacious ventricle presents the rudiment of a septum ventriculorum, which becomes so far developed as finally to exhibit only an aperture which is usually situated at its upper extremity. The most common anomaly of the vascular system combined with this form is the origin of the aorta from both ventricles, and the displacement of the pulmonary artery. The foramen ovale in the partition between the auricles remains open. In other cases the septum is perfect, but so situated as considerably to diminish the size of one or other of the ventricles, interfering with its valvular apparatus, and giving the auriculo-ventricular opening a very contracted and even closed appearance,—a condition of things that involves the patency of the foramen ovale and of the ductus arteriosus.

e. Here we have a form of the heart in which the partition between the auricles is defective, although there is a perfect separation of the ventricles. The degree and form of this defective structure are very variable. The septum is sometimes entirely absent, its line of direction being simply indicated by several soft membranous filaments which pass from the posterior to the anterior wall of the common cavity of the auricles. In other cases the rudiment of a septum atriorum developes itself in the form of a crescentic band, either from the arch of the auricle, or below the septum ventriculorum. The wide aperture of communication between the two auricles is round or oval, and has its major axis inclined from before backwards. In other cases, the rudimentary structure is sometimes so far developed round the septum that this deficiency is often represented by a smaller and obtuse triangular aperture; and in other cases, again, the septum seems so far developed from above that it may easily contain a foramen ovale. There are, in this case, two apertures in the partition between the auricles, the former, which depends on defective formation, is not closed, and the latter (the foramen ovale) remains open.

Cases of this nature are generally characterized by a congenital contraction or insufficiency of the aorta, by extraordinary dilatation of the pulmonary artery, and by eccentric hypertrophy of the right side of the heart.

f. In this form the fœtal passages,—the foramen ovale, and the ductus arteriosus,—remain open. The degree of the patency of the foramen ovale varies considerably, its valve being very nearly or entirely absent in some cases, but more commonly the upper third or fourth portion of it is wanting, and most frequently of all there is a mere deficiency of attachment at the upper part of the isthmus, by which means a fissure rather than a foramen is formed, which communicates in a very oblique direction from below and behind upwards and forwards from the right into the left auricle. This fœtal condition is sometimes persistent to a greater or less degree, and consists in this—that under a marginal projecting rudiment of the Eustachian valve, which penetrates into the anterior columna isthmi fossæ ovalis, there is a communication with this fissure-like aperture, or with the still patent foramen ovale. The opening at the upper boundary of the isthmus is either formed as a simple fissure, or consists of several small and roundish apertures. The cause of the patency of the foramen ovale frequently depends on the different malformations of the heart already enumerated, and on the different anomalies of the arterial vascular trunks and of the ductus arteriosus, which still remain to be noticed. The patency of the foramen ovale most frequently corresponds with an incidental arrest of development. In some cases it is associated with smallness of the heart, and retraction of the apex (the fœtal condition). It is of very frequent occurrence in its lesser degrees.

The patency of the ductus arteriosus will be more fully noticed in a future page.

The following are the most important of these *Anomalies of the Vascular Trunks:*

1. Those affecting the *Aorta.*

a. There may be a single arterial trunk, which may be regarded as an aorta sending off branches from different points to the lungs.

b. The aorta may be a vessel from which only the branches of the upper half of the body (and not all of these) are given off, whilst the pulmonary artery, through the ductus arteriosus, constitutes the descending aorta.

c. There may be different degrees of obstruction of the aorta, which may be either very narrow or quite closed from its origin to its point of junction with the ductus arteriosus, in which case it is supplied by the latter with blood from the pulmonary artery. The whole arterial trunk with its ramifications,—in short the whole arterial system,—is in this case frequently disproportionally narrow.

d. The aorta may originate from both ventricles, owing to a deficiency in the partition-wall between them, in which case it is deflected somewhat to the right. The pulmonary artery is in this case, either normal, or, as is frequently observed, it is obstructed, narrow, and even closed. The ductus arteriosus, if it be present, then remains open, and carries the aortic blood to the pulmonary artery.

2. Those affecting the *Pulmonary Artery*.

a. The trunk of the pulmonary artery is not only absent where the lungs are wanting, but even where these are present and are furnished with vessels from the aorta.

b. Obstruction of the pulmonary artery, which may be either too narrow or wholly closed, in which case the blood is conveyed to it through the ductus arteriosus from the aorta. This occurs when the right ventricle is imperfect, when the conus arteriosus ends in a *cul de sac*, and very commonly when the aorta originates from both ventricles.

To the above we may add—

3. Anomalies of the *Ductus Arteriosus*.

a. It is sometimes wholly absent.

b. Besides sending several vessels to the head and upper extremities, as do also the branches of the pulmonary artery, it supplies the aorta descendens, or rather merges into it. In this case the aorta diminishes in calibre after giving off its branches, and merges as a thin vessel into the large ductus arteriosus after the latter has been curved into an aorta descendens (Kilian). This anomaly occurs either separately and independently, or conjointly with other anomalies of the vascular trunks and of the heart.

c. The most common anomaly is a defective involution of the ductus arteriosus after birth; it either remains open or even in some cases experiences a dilatation. It is, however, much more rarely open than the foramen ovale, whose patency is in general a necessary consequence of different anomalies of the heart and vascular trunks, although even here many exceptions present themselves. On the one hand, it may remain open without any palpable anomaly of the heart and vascular trunks, and on the other it may be contracted, and even closed, where such anomalies exist.

Its involution may be hindered either by the pulmonary artery or the aorta; thus it may either be entirely patent, or may exist as a mere opening closed in the direction of the pulmonary artery, but patent towards the aorta. To this class belong those cases in adults, where the ductus arteriosus forms a sac-like appendage to the aorta, and where the obliterated ostium arteriæ pulmonalis has the appearance of having been reopened by violence.

When the ductus arteriosus remains patent, many causes combine to keep the foramen ovale open.

Finally, we also observe a deficiency in the formation of the *valves*. The auriculo-ventricular valves present various obstructions and malformations, generally in connection with a simultaneous malformation of the corresponding ventricle, or associated with a contraction or occlusion of the opening, and principally on the right side. In reference to the arterial valves those of the pulmonary artery are occasionally absent, while malformation and closure of the latter artery, a blind termination of the conus arteriosus, and an abnormal condition of the whole of the right ventricle, are simultaneously present. These valves not unfrequently assume an abnormally inflated annular form, and in some cases only two instead of three valves are observed in the pulmonary artery or in the aorta.

The valve of the foramen ovale is either wholly absent or, as we have already remarked, is imperfect in various ways. In rare cases the Eustachian valve has been found wanting. Its defective involution after birth is interesting, inasmuch as it gives rise to the imperfect closure of the foramen ovale. The Thebesian valve has been found wanting in a few cases.

Malformations *per excessum* affect the heart and vascular trunks in various modes and degrees, and may be referred either to a duplication, or to an arrest of development.

To the *former* belong—

Complete duplication of the heart, or the occurrence of two separate hearts in two distinct pericardia, or in one common pericardium, which is not unfrequent in double monsters, especially where there is duplication of the upper half of the body, while the observations on record regarding a double heart in a single body, are very few in number, and of doubtful character.

Duplication of one or more portions of the heart, or the presence of supernumerary, more or less perfectly separated cavities from which numerous vessels proceed, as if of very rare occurrence when there is only a single body. We observe, however, occasionally, in normally formed individuals, a rudimentary partition projecting, in the form of a band or amorphous mass of muscle, into one of the cavities of the heart.

A large heart, from which proceed double vessels, is found in double monsters.

To the *latter* belong—

The persistence of a double aorta ascendens, a cleft condition of that vessel, the persistence of a ductus arteriosus on the right side (Breschet), and the duplication of the upper as well as the lower vena cava.

The valves also occasionally exhibit an excess of formation; we have either an increase of the apices of the auriculo-ventricular valve of the right side, or supernumerary valves, or a multiplication of their apices, with perforation of the ventricular partition; or there may be four semilunar valves in the aorta or the pulmonary artery, or duplication of the Thebesian valve, &c.

§ 2. *Anomalies of Form.*

These are of comparatively common occurrence, and may affect either the external form of the heart, or its internal arrangement; occasionally both varieties are simultaneously present, in which case the deviation from the normal type in the external form is dependent on the anomaly in the internal structure. These anomalies are, moreover, either *congenital* or acquired, the former comprising, more especially, original malformations depending upon arrest of development; and the latter, the numerous and various deviations of form developed at different periods of life, and even in the fœtus, as consecutive anomalies, arising more particularly from hypertrophy and dilatation.

The most important original anomalies in the external form of the heart, are combined with and dependent on the already described important anomalies of the internal structure and of the vessels; the more unimportant may be present, associated with very trifling internal anomalies, as for instance, the patency of the foramen ovale, or even, where none of these exist, and where the internal structure is perfectly normal. To this class belong the retraction of the heart's apex—apex cordis bifidus, an arrest at an early stage of development—and the rounding of the apex of the heart, associated with predominant width of the whole heart, an arrest at a very advanced period of development.

This last-named form, which depends on an equality in the size and thickness of both ventricles, is often continued to late periods of extra-uterine existence, and is maintained, together with the simultaneous patency of the fœtal passages, by the defective development of the lungs, whose proper functions may be mechanically obstructed by the form of the sternum, as is observed in rachitis.

Among the unimportant and incidental anomalies depending, very frequently, on various degrees of contraction and rigor, we must reckon different forms of the heart which aproximate to the round type, and are either long, slender, wedge-shaped, pad-like, spirally curved, broad and obtuse, &c.

In order to avoid repetition, we would refer our readers to the sections on hypertrophies, dilatations, and textural diseases, for further and special notice of the *acquired* anomalies of the heart.

§ 3. *Anomalies of Position.*

These are either *congenital and original*, or *acquired.* The *former* are very numerous, and admit, in part, of being referred to an arrest of development. Many depend on different adhesions of the heart resulting from inflammation in the fœtus, and some again on different anomalies of neighboring organs, as, for instance, on the deficient development of a lung, the partial deficiency of the diaphragm, and the position of the abdominal viscera in the thorax. These anomalies are very various in their character, and the most important, together with the vascular anomalies included in this case, are as follows:

Position of the Heart exterior to the Body.—This anomaly occurs associated with a partial absence of the diaphragm and the abdominal and thoracic walls. Where the former of these is absent, the heart is generally situated with all or several of the viscera externally to the body, in a closed or open sac occasionally contained in the sheath of the umbilical vessels.

Position of the Heart within the Body, but external to the Thoracic Cavity.—According to the direction in which the heart is placed, it assumes either a cervical position (*ectopie cephalique*), or an abdominal one (Breschet).

Anomalous Positions of the Heart in the Thoracic Cavity.—These possess various points of interest from their presenting considerable analogy with many acquired anomalous positions of the heart, and also on account of their apparently arising from similar conditions. To these belong the position of the heart on the right side, without the simultaneous transposition of other viscera, its perpendicular position in the centre of the thoracic cavity, its horizontal, oblique positions, &c.

Anomalous Origin of the Vascular trunks.—To this class belong:

The displacement of the aorta towards the right side, and its origin from both ventricles, associated with a defect in the ventricular septum; or the aorta may take its origin, conjointly with the pulmonary artery, from the right ventricle, where no such anomaly exists. We sometimes find a similar relation of the pulmonary artery, that is to say, it takes its origin from both ventricles, or conjointly with the aorta from the left ventricle. This vessel has also been observed to spring from abnormal positions in the right ventricle.

Many anomalies of the systemic and pulmonary veins, as, for instance, the opening of a left descending vena cava into the auricle, an opening of the pulmonary veins of the right side into the right auricle, into the upper vena cava, &c., also belong to this class.

Actual transposition may exist with reference to the heart alone, or conjointly with the thoracic and abdominal viscera generally. A more important transposition is, however, that affecting the vascular trunks, which often present the anomaly of the aorta, springing from the right, and the pulmonary artery from the left ventricle, while the veins open normally. Otto found, in the case of a double monster, that the venæ cavæ opened into the left, and the pulmonary veins into the right auricle, while the arterial trunks presented the normal mode of origin.

The acquired changes of position of the heart are very numerous, but as they are merely secondary phenomena, they generally possess a very subordinate interest. An exception occurs, however, in the case of those anomalies of position of the heart, which arise from empyema, pneumothorax, pulmonary emphysema, atrophy of the lungs, &c., and which are of great importance with regard to diagnosis.

The majority of these consist in a displacement of the heart from its normal position. It may occur in the most opposite inclinations either to one or the other side, or downwards, upwards, forwards, or backwards. The most common causes of these displacements are, on the one hand, excessive dilatation of one or other of the pleural sacs from exudations into its cavity, from pneumothorax, or pulmonary emphysema; and on the other, the formation of a vacuum in it by the cure of chronic pleurisies, by wasting and atrophy of the lungs consequent on indurated pneumonia, by bronchial dilatation, &c. The displacements of the heart towards one or the other side resemble similar congenital and original anomalies of the heart's position. A change in the position of the heart is but rarely occasioned by pneumonic and tuberculous enlargements of a lung, and still more unfrequently by an acquired position of the abdominal viscera in one side of the thorax, arising from laceration of the diaphragm, &c. The heart may likewise be differently displaced from its position by aneurisms of the aorta, the contiguity of voluminous adventitious products, &c. Flatulence, extreme ascites, and large adventitious products in the abdomen, may also displace the heart in an upward direction; whilst a corresponding anomaly in the position of the heart is likewise induced by curvature of the spine, irregularity in the form of the thorax, &c.

In contrast with the above-named anomalies, this change of position may sometimes be *spontaneous*, in consequence of the heart assuming an anomalous position and anomalous relations of contact with the diaphragm and ribs, arising from an uniform or a varying enlargement, and from its simultaneous increase in weight.

§ 4. *Anomalies of Size.*

These anomalies manifest themselves either by *an abnormal excess* or *deficiency of size.* Both conditions may be either congenital or acquired, and are of great importance from their frequent occurrence and the serious and numerous consecutive disturbances to which they give rise.

In order to arrive at a correct opinion regarding an individual case, it is especially necessary for the student to acquire a knowledge of the normal size of the heart and of its individual portions. Many measurements have been made in recent times, and from these a mean or average standard has been deduced.

The results yielded by Bizot's measurements appear to us to be most correct. We will limit ourselves to the dimensions in adults, between the ages of 30 and 49 years.

The following are the mean measurements:

	IN MEN. Paris lines.	IN WOMEN. Paris lines.
The length of the heart, ...	$43\frac{3}{23}$	$41\frac{2}{27}$
The breadth "	$47\frac{18}{23}$	$44\frac{1}{27}$
The thickness "	$17\frac{4}{23}$	$14\frac{4}{27}$
The length of the left ventricle, ...	$29\frac{11}{23}$	$31\frac{16}{17}$
The breadth " "	$53\frac{4}{23}$	$46\frac{4}{17}$
The length of the right ventricle, ...	$37\frac{13}{23}$	$33\frac{13}{27}$
The breadth " "	$83\frac{10}{23}$	$76\frac{17}{27}$
The thickness of the walls of the left ventricle:		
" " " at the base, . .	$4\frac{17}{46}$	$4\frac{1}{6}$
" " " in the middle, . .	$5\frac{1}{11}$	$4\frac{37}{54}$
" " " near the apex, . .	$3\frac{13}{23}$	$3\frac{6}{27}$
The thickness of the interventricular septum in the centre, ...	$4\frac{21}{23}$	$4\frac{11}{27}$
The thickness of the walls of the right ventricle:		
" " " at the base, . .	$1\frac{39}{46}$	$1\frac{19}{27}$
" " " in the middle, . .	$1\frac{7}{23}$	$1\frac{13}{54}$
" " " near the apex, . .	$\frac{45}{46}$	$\frac{26}{27}$
The width of the auriculo-ventricular openings:		
" " " of the left, . .	$48\frac{9}{22}$	$40\frac{17}{26}$
" " " of the right, . .	$54\frac{6}{23}$	$47\frac{4}{27}$
The width of the aorta (above the valves), . .	$30\frac{20}{23}$	$28\frac{3}{27}$
" " origin of the pulmonary artery, .	$31\frac{13}{23}$	$29\frac{1}{5}$

We purpose, in the sequel, making further use of the other results of the labors of Bizot, which possess any degree of interest, and are not opposed to our own observations.

According to Bizot, the heart increases in volume from birth to extreme age; this increase being most considerable to the age of 29, after which it is only appreciable by measurement. Augmentation of volume depends especially on the continuous dilatation of the openings, and on the increase of thickness of the walls of the ventricles, which is always most strongly marked in the case of the left, and is indeed scarcely perceptible in that of the right ventricle. The dilatation of the auriculo-ventricular openings is tolerably uniform, and that of the arterial equally so until middle life, but after that period the opening of the aorta is more rapidly dilated than that of the pulmonary artery, the latter becoming even narrower than the aorta. In children both the arterial openings remain equally wide, till from the sixth to the tenth year. The cavities of the right side of the heart have a greater capacity, and their openings are wider.

Bizot's opinions regarding the influence of sex and bodily frame are, that the dimensions of all the parts collectively are smaller in women than in men; that the auriculo-ventricular openings in particular are narrower whilst the opening of the pulmonary artery is relatively wider in them than in men. In tall persons of either sex, the heart, according to the same authority, is relatively smaller than in persons of shorter stature, while it is larger in broad than in narrow-shouldered persons.

We adopt Bouillaud's data for the walls of the auricles, with a remark, however, that his estimate is too high; according to him, the thickness of the wall of the left auricle is one Paris line and a half, while that of the right auricle is one line.

Laennec proposed to establish a scale for the relative measurement of the size of the heart, and rejected as inefficient all data of the weight and size of the heart that had been obtained without regard to individual bulk. The basis on which he founded his conclusions was, that the heart, including the auricular appendages, should be of a volume equal to that of the fist of the individual, or only in a slight degree either larger or smaller. The walls of the left ventricle should be somewhat more than twice as thick as those of the right one; the left ventricle, when cut open, should remain unclosed, while the somewhat wider right ventricle, which, notwithstanding the thinness of its walls, is furnished with more considerable trabeculæ, should collapse.

If in the consideration of these data (which are, however, in many respects defective), regarding the relative thickness of the walls of the right and left ventricles, we bear in mind that this relation is not only very commonly, according to Andral's observation, as 1 : 3, but still more frequently (see Bizot's data), as 1 : 4,[1] and that these measurements refer only to middle life, we shall find that Laennec's comparison of the size of the heart with that of the fist deserves considerable attention, remembering that it must be received as simply approximative, and limits itself to cases where there is no apparent disproportion in the size of the fist. We have, therefore, as a general rule, regarded the heart as of a relatively normal size, when it was equal to that of the fist, and when there was an absence, both during life and after death, of any indications of cardiac disease.

The weight of the heart, in its normal condition, has been variously estimated, and may be from eight to ten ounces. (Compare Lobstein, Bouillaud, and Cruveilhier.)

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[1] In all these statements the columnæ carnæ are not taken into consideration.

A. *Abnormal Size.*

Deviations in the size of the heart depend either on *hypertrophy* of its muscular substance (augmentation of its mass) or on *dilatation* of its cavities; while both conditions, with a preponderance of the one or the other, very commonly constitute the basis of the higher degrees of enlargement of the heart, as we shall presently have occasion to show.

Here, as has already been indicated, we will merely consider hypertrophy of the muscular substance. There is, however, a form of disease, for which we know no better denomination than *hypertrophy of the endocardium*, and of which, however much it may seem to belong to the subject under consideration, we will treat subsequently in connection with endocarditis, a disease with whose products it may easily be confounded, and for which it undoubtedly is frequently mistaken. We will also defer the consideration of *hypertrophy of the valves* to a subsequent portion of our work.

a. Hypertrophy of the muscular substance of the heart (*hypertrophia cordis*) constitutes either *total* or *partial hypertrophy*, as it affects the whole, or only some portions of the heart, and is characterized by various degrees of intensity.

Total hypertrophy is, in most cases, so far unequal that it usually preponderates in one section of the heart,—commonly, although not invariably, in the left portion,—where it forms the starting-point of a morbid development of bulk.

Partial hypertrophy affects either the whole of one of the larger sections of the heart, as for instance the walls of one of the cavities, or it attacks only certain parts of that section. Thus it is very frequently limited to the true muscular wall of one ventricle, thickening it in various degrees, while the papillary muscles and the trabeculæ retain their normal volume, or even where they have become perceptibly thinner and fainter simultaneously with the dilatation of the affected cavity. In other cases hypertrophy principally attacks the papillary muscles and the trabeculæ, whilst the true fleshy wall is only moderately increased in thickness. The former of these conditions occurs generally in the left; and the latter, that is to say, an excessive development of the trabeculæ, in the right ventricle.

Hypertrophy of the muscular wall of a ventricle may present great variations; it may, in one case, affect the whole, in another it may be limited to one portion of the ventricle, as, for instance the base, the middle part or the septum, or again it may predominate in one or other of those sections.

Hypertrophy of the auricles is generally uniformly diffused over the wall, but it occasionally preponderates in the atrium towards the appendage.

The degrees of hypertrophy present still more numerous differences. If we follow Bizot's data we must assume that ventricular hypertrophy is present when the thickness of the muscular wall of the left ventricle in men is about 6′′′ (Paris measure), and in women is about 5′′′, and when the right ventricle in men is about 3′′′ in thickness, and that in women 2½′′′ in thickness. From this point hypertrophy may pass through every varying degree, till it induces so enormous an increase of bulk, that the walls of the left ventricle attain a thickness, varying from an inch to an inch and a half; that the walls of the right ventricle vary from 6 to 9 lines, that the walls of the left auricle vary from 2 to 3 lines, or even more, and that the walls of the right auricle vary from 1½ to 2 lines.

The weight of an hypertrophied heart may range from one to two pounds, and even higher.

The most important and serviceable classification of hypertrophies of the heart is that which is based on a reference to the condition of the cavities of the heart, more especially in regard to their capacity (Bertin, Bouillaud).

1. The capacity of the hypertrophied portion of the heart may remain normal, constituting *simple hypertrophy*, in which the dimensions of the heart are increased.

2. The cavity of the heart may be dilated, constituting *eccentric hypertrophy* (*hypertrophia excentrica, centrifuga*); here also the dimensions of the heart are increased.

3. The cavity of the heart may be contracted, constituting *concentric hypertrophy* (*hypertrophia concentrica, centripeta*). The dimensions of the heart may here be increased, normal or diminished.

We shall treat more fully of these different forms when we take into consideration the most essential points relating to dilatation of the cavities of the heart.

b. The dimensions of the heart are more increased by the dilatation of its cavities than by hypertrophy. This dilatation (*dilatatio cordis*, also *aneurysma cordis* in the old writers), may, like hypertrophy, be *total*, affecting all the cavities of the heart, or *partial*, attacking only one of these portions. The excessive degree which dilatation may attain led the early anatomists to compare the human heart to that of a bullock.

Here also the most useful classification is that which is founded on the relation of the walls of the diseased portions of the heart.

1. Dilatation of the cavities of the heart may occur conjointly with hypertrophy of their walls, constituting *aneurysma cordis activum* (Corvisart), and with *eccentric hypertrophy*.

2. Dilatation of the cavities of the heart may exist, associated with walls of normal thickness. This condition of simple dilatation—*aneurysma cordis simplex*—deserves equal attention with the abovenamed form of *dilatation with hypertrophy*, in as far as the normal thickness of the walls of the heart in dilatation of the cavities must necessarily depend on hypertrophy; it may therefore be regarded as an *active* dilatation—*aneurysma cordis activum*.

3. Dilatation of the cavities of the heart may occur in combination with attenuation and relaxation of the walls, constituting *passive* dilatation—*aneurysma cordis passivum* (Corvisart).

Besides these forms of dilatation, there are others connected with and dependent on alterations of texture, limited on one portion of a single cavity, and which we will pass over in the present case, reserving their consideration for a more suitable occasion, under the head of inflammations of the heart. We would here merely observe, that these forms of dilatation are known as *partial dilatation* and *partial aneurism of the heart*. As, however, we have applied the former of these terms to dilatation of a single division of the heart, and as, on the one hand, the term aneurism is unsuitable to the forms of dilatation under consideration, while, on the other, dilatation, combined with and dependent on alterations of texture, and such as attack only one portion of a cavity of the heart, exhibit great affinity with aneurisms of the arteries, we would designate these last named, which we pass over for the present, as true *aneurism of the heart*.

On considering the above-named forms of hypertrophy and dilatation, we find *five*, or perhaps more correctly speaking, *four different conditions*, and these will constitute the subject of the following remarks.

Simple hypertrophy, which is in general of rare occurrence, affects the ventricles, attacking the left one more frequently than the right. It probably continues to exist only for a certain period of indefinite duration, and then gradually merges into eccentric hypertrophy, that is to say, hypertrophy with dilatation. Although scarcely a doubt can be entertained of its existence, the attempt to confirm it is not devoid of difficulty.

The existence of *concentric hypertrophy* has been doubted by many who have made observations on the human subject after death, and prosecuted experiments on animals. In those who die from loss of blood, and occasionally after sudden and violent modes of death, the heart is indeed often in a condition of contraction, which might easily be mistaken for concentric hypertrophy. We cannot, however, agree with those who doubt the existence of concentric hypertrophy, which occurs, although rarely, in both ventricles, and, according to our observations, more frequently in the left. The cavity of this portion of the heart appears contracted in consequence of the thickening of the muscular wall, and of the papillary muscles, and the trabeculæ. Disease of the heart is manifested during life, and the symptoms exhibited correspond to the appearances observed after death.

Eccentric hypertrophy, *active dilatation*, including *simple dilatation*, is incomparably the most frequent condition. It attacks the ventricles as well as the auricles, and most frequently the cavities of the left side. Active dilatations originate in one portion of the heart, and, beginning at the left ventricle, gradually extend over the whole organ. This condition gives rise to the highest degrees of cardiac enlargement, which were known to the ancients under the terms *enormitas cordis*, *cor taurinum*, &c. The enlargement is most strikingly manifested at the conus arteriosus of the right ventricle, whilst the space of the actual ventricle is generally contracted by the intrusion of the arch of the septum. The auricles are occasionally the special seat of active dilatation, and in these cases the disease commonly depends on contraction of the auriculo-ventricular opening on the same side. The wall of the auricle is stiff and rigid, and the cavity is not unfrequently filled with coagulated blood, or occasionally with stratified coagula of fibrin. These forms of dilatation have, however, been observed unassociated with this form of contraction.

Passive dilatation, in its lesser degrees, is of frequent occurrence. It attacks the ventricles as well as the auricles, especially the cavities on the right side of the heart, and the right auricle most frequently. When it affects the left ventricle it is most commonly and most decidedly seen at its apex, where it first manifests itself. Intense degrees of this form of disease are unusual, although the auricles in particular are capable of remarkable dilatation.

These different forms may be variously combined. Where disease has attacked the whole of the cavities of the heart, one cavity is usually disproportionally affected beyond the others, and heterogeneous forms are observed to arise and exist in conjunction with one another, as for instance hypertrophy and active dilatation on the left side, together with passive dilatation of the right. In most cases the disease predominates in that portion of the heart's cavity which was first and, from some obvious cause, most intensely affected. Such is, however, not invariably the case, since consecutive disease occasionally supervenes, which fully equals or even exceeds that in the cavity originally attacked.

Dilatation of the openings usually exists in conjunction with dilatations of the heart generally, corresponding in intensity with the various degrees of the latter, and depending most probably on one common cause. In active dilatations, the arterial openings are more prominently affected, whilst in passive dilatation the auriculo-ventricular openings more frequently participate in the disease. In this form of dilatation the valvular apparatus very commonly remains sufficient, in consequence of an enlargement of the valves, attended by a striking attenuation and an elongation of the tendons of the papillary muscles.—We must, however, be careful not to confound these forms of dilatation with dilatation of the commencement of the aorta, which is of very frequent occurrence, and depends on a diseased condition of its coats, for the latter will occasion dilatation of the left side of the heart, with a frequency proportional to the association of dilatation of the vessel with insufficiency of its valves.

It is important to notice that there is a relaxed condition of the heart after death, which is very similar to *passive dilatation.* In the rapidly decomposing bodies of those who have died of acute dyscrasiæ, the heart is very commonly collapsed, visibly dilated, easily torn, and characterized by thinness of the walls, various discolorations of the muscular substance, and imbibition of hæmatin in the endocardium and along the coronary veins. It is very probable that a similar condition of the heart manifests itself in every case at a certain period after death. The above-named requirements of its occurrence enable us to recognize this phenomenon as the result of decomposition, but the difficulty attending its diagnosis in the dead body reminds us of that which attaches to the question of the existence of concentric hypertrophy in so far as this condition is undoubtedly very frequently to be referred to the *agonia mortis.*

In *simple dilatation* we also occasionally meet with a condition of the muscular substance of the heart, which gives it a passive character.

In cases of *active dilatation* (*eccentric hypertrophy*) the trabeculæ are frequently so completely atrophied as not only to be attenuated by elongation, but even entirely severed, their existence being indicated along the greater and middle part merely by the inner cardiac investment surrounding them, and by the muscular substance of which their terminations are composed.

We are not yet able to explain why, under analogous or very similar conditions, dilatation of the heart will be developed in one case in a passive, and in another in an active form. We will append to our enumeration of the causes of these diseases of the heart the form of disease that is usually dependent on each, merely remarking here, in general terms, that, in our opinion, considerable mechanical obstructions generally, and sometimes with great rapidity, induce an excessive degree of dilatation, whilst, on the other hand, lesser and more slowly developed obstructions give rise to hypertrophy.

The *form* of the heart undergoes various alterations in consequence of these enlargements. Its malformation is the more important in proportion to the enlargement, and the more it is confined to, or preponderates in one single cavity of the heart. It affects the external as well as the internal form. In simple, and still more manifestly in eccentric hypertrophy (active dilatation) of the left ventricle, where the chief seat of disease is at the base and the middle portion, the heart assumes a round wedge-like form, while in the more advanced stages of the disease, the whole ventricle is swelled into a pad-like shape. The malformation which especially consists in dilatations of the left ventricle, expands towards the right ventricle, into which the septum is bent in an arched form; its space being so considerably contracted, that it appears like a mere appendix to the heart, while its conus arteriosus appears dilated and hypertrophied. Dilatations of the right ventricle widen the heart at its base, and from thence down to the apex. Where there is simultaneous dilatation of the left ventricle, the heart acquires the form of an obtuse triangular pyramid, or a discoidal mass. Active dilatations of the conus arteriosus of the right ventricle, which are of frequent occurrence, lead to malformation of the heart by enlarging its circumference near the base, &c.

The *position* of the diseased heart becomes the more anomalous in proportion to the volume and weight which it acquires. In a slight degree of enlargement, the heart inclines less to the left side of the thorax, while in excessive forms of enlargement and dilatation, it has its base almost diagonally inclined to the right and its apex to the left side, whilst its right half rests on the anterior thoracic wall, contracting both thoracic cavities in the region of the lower lobes of the lungs, and causing them to press in one large surface on the diaphragm, which is thus more or less pushed downwards on the epigastrium.

The *color*, *consistence*, and *texture* of the muscular substance of the diseased heart, present numerous differences.

The color of the *hypertrophied heart* is most frequently dark, and of a brownish-red hue; the consistence is generally greatly increased, and the texture apparently normal. It must here be remarked that the consistence of the right ventricle presents a striking anomaly in the more highly developed forms of hypertrophy, the texture acquiring a toughness which is never observed under any condition in the left ventricle. The walls which become rigid and retract on being cut, exhibit extreme resistance and hardness, and yield, when struck, a sound which, according to Laennec, resembles the tone emitted from hard leather. A similar relation is observed in active dilatation of the auricles, when excessively hypertrophied. This increase of consistence seems to depend on the deposition of a great quantity of organic matter in the form of a finely granular substance, and in the production of new flat muscular fibres without transverse striæ.

In other, and very frequent cases, the hypertrophied tissue of the left ventricle presents another character. Its color appears to be faded, and of a dirty brown or yellow tint, either in separate points in the form of foci, or over a layer, generally an internal one, whose thickness varies, or finally throughout the whole thickness of this portion of the heart. The consistence then becomes modified in a peculiar manner, the walls of the heart become rigid, tough, and capable of resistance, while their tissue loses its proper firmness, is fragile, and easily broken down. The texture is perceptibly altered, although in what manner the change is effected is not known. According to our investigations, this disease of texture must be regarded as a form of morbid fatty degeneration of the heart, similar to that treated of under Form 2 of Diseases of the Muscles; and we will therefore consider the subject more at large under the head of Anomalies of Texture. We are moreover of opinion, that it associates itself with hypertrophy as a consecutive disease; that is to say, that after being once developed in the hypertrophied tissue, it favors the dilatation of the hypertrophied portion of the heart, and very frequently gives rise to those spontaneous ruptures which occur in this organ.

Traces of inflammation not unfrequently occur, either with or without the above-named alterations of texture in the muscular substances of the left ventricle, when it is the seat of active dilatation. One or more points or foci of limited extent, either on the surface or lying deep in the texture, occasionally exhibit a redness and injection of the bleached and flabby tissue, which is infiltrated with gelatinous, fibrinous or purulent matter. More frequently these are the residua of a former inflammation,—spots at which we find the muscular substance replaced by a white ligamentous (fibroid) texture. (See the section on "Inflammation of the Muscular Substance of the Heart.") These latent and recurring processes of inflammation are in some instances connected with the residua and secondary effects of pericarditis and endocarditis, and undoubtedly would appear to promote the origin and further development of cardiac disease.

In *passive dilatations*, the color of the tissue of the heart is occasionally purplish-red, but more frequently it is darker and bluish-red, owing to the imbibition of the hæmatin, which is greatly favored by the dissolved condition of the blood, and the relaxation of the whole tissue. The muscular substance of the heart is in these cases extremely flabby and easily torn, while its walls collapse when they are cut open. In the higher stages of dilatation the muscular bundles in the auricles are forced asunder, so that the wall of the heart appears between them as a mere membrane.

In dilatations arising in pericarditis, the muscular substance has a dirty rusty brown, or yellow leather-like color, is easily torn, and appears as if half boiled; in other cases, it is pale, flabby, and abnormally fat, the surface of the heart being frequently covered by an accumulation of adipose matter.

The knowledge of the *causes of the origin of these diseases of the heart* is of the highest interest. Many admit of being discovered and made apparent without any great difficulty, but many others are partly problematical and partly uninvestigated. We will consider them in such an order as to proceed from those which are obvious to those which are less apparent, and finally to the problematical and hypothetical, giving special attention to the practically important ones comprised under each category.

These causes are as follow:

1. *Mechanical Obstructions*, which give rise, according to circumstances, either to preponderance of dilatation or preponderance of hypertrophy.

a. Mechanical Obstructions in the Ostia of the Heart.—The number of diseases produced by these causes is probably the greatest. They are consequent on various diseases of the arterial and auriculo-ventricular valves, more especially on the secondary effects of endocarditis (Bouillaud's chronic endocarditis), and admit of being generally referred to *contraction* (*stenosis*) *of the ostium*, and to *insufficiency of its valves.* Whether the latter has a tendency to give rise more frequently to dilatation, and the former to hypertrophy, has not yet been determined.

As these diseases of the valves are far more frequent in the left than in the right side of the heart, so also are the diseases of the heart to which they give rise. The auricle or the ventricle becomes the more acutely affected according to the seat of the valvular disease, while both are simultaneously attacked where the arterial and auriculo-ventricular valves are alike diseased. Owing to the impediment presented to the emptying of the heart's cavities on the left side, and the consequent obstruction of the capillary circulation through the lungs, disease extends to the right ventricle, and from thence to the right auricle, commonly manifesting itself as hypertrophy with excessive dilatation, and occasionally—more especially in the auricle—as passive dilatation.

b. Mechanical Obstructions in the Arterial Trunks, occurring at different distances from the heart, promote the development of cardiac disease in proportion to their greater vicinity to that organ.

These obstructions are of various kinds.

Congenital contraction of one or other of the vascular trunks is not unfrequent, and is generally manifested in the aortic trunk by an insufficiency of calibre which extends to the branches, and probably also to the more delicate ramifications. This condition induces very considerable dilatation with more or less hypertrophy in the left ventricle, and subsequently leads to similar dilatations of the left auricle, and of the cavities on the right side of the heart.

To this class belong acquired contractions of the vascular trunks and their main branches, together with their final obliteration, dependent on alterations of texture.

Further, the contractions caused by compression or expansion the obstructions presented to the current of the blood by the angular curvature, twisting, &c., of the large arteries.

Lastly, there are also dilatations of these arterial trunks, which, as is well known, appear frequently, and in the most excessive degrees, in the trunk of the aorta and its main branches, under the various forms of aneurism. These conditions generally induce active dilatation of the left ventricle, with a rapidity and intensity proportional to their vicinity to the heart and to their importance. It must be observed, that although insufficiency of the valves of the diseased trunk may simultaneously occur, its existence is not necessary to the formation of cardiac disease, which is then owing to the obstacle opposed to the advance of each successive blood-wave by the mass of blood accumulated in the dilated and paralyzed trunk.

c. Similar (mechanical) Obstructions in the Capillaries.—Obstructions in the capillaries of the pulmonary artery are tolerably evident, inducing active dilatations of the right ventricle, and, subsequently, dilatation of the right auricle, having in some cases a more active and in others a more passive character. To these belong:

α. The obstruction presented to the circulation through the capillary system of the lungs by contraction of the thoracic cavity, and the consequent excessive thickness of the pulmonary texture. This condition presents a great degree of intensity and a character of constancy in malformations of the thorax and contractions of its cavities consequent on curvature of the spine, more especially extreme scoliosis and kyphosis, and in cases of rachitic chicken breast. When the above-named diseases of the right side of the heart attain a great degree of intensity, they commonly give rise to highly developed hypertrophy.

β. Next, we must notice an increased condensation and atrophy in a more or less considerable portion of the lung, in consequence of compression from pleuritic exudation and of its healing, and of indurated pneumonia. The development of the heart will be proportional to the actual atrophy of the pulmonary texture, to the extent of the surface atrophied, and to the degree in which the capillaries have been destroyed by obliteration.

γ. Atrophy of the pulmonary texture associated with extended and considerable bronchial dilatation.

δ. The obstruction presented to the circulation through the capillaries of the lungs by their emphysema (emphysema vesiculare). This condition depends at first on the continued excessive expansion of the pulmonary cells, and in the more advanced stages on the obliteration of the capillaries which occurs in an uniform degree with the atrophy of the texture of the lung. The importance of the heart-disease depends on the extent, degree, and period of duration of the emphysema; occasionally the disease is very intense.

ε. The obstruction opposed to the injection of the lungs by the pulmonary artery, owing to the insufficient emptying of the pulmonary veins in consequence of disease of the left side of the heart, and of the habitual over-filled condition of the capillaries of the lungs. Hence arise the greatest number of diseases of the right side of the heart, and all the numerous consecutively developed dilatations of the right ventricle and auricle, which originate in the left cavities, and are extended by means of the capillaries of the pulmonary system. The most important of these forms of dilatation are those which, in the manner already indicated, depend upon a contraction of the auriculo-ventricular opening on the left side of the heart.

It is only in comparatively rare cases that tuberculosis of the lungs and tuberculous pulmonary phthisis give rise to even a very moderate degree of active dilatation of the right side of the heart. A certain degree of diminution in the size of the heart is more frequent, and is then manifested in the form of simple or even concentric atrophy, which corresponds with the general tabes and the wasting of the mass of the blood.

On the other hand, similar obstructions in the *capillaries of the aortic system* are either wholly unknown, or are so obscure, that although *à priori* conjectures may be hazarded regarding their persistent or transient existence, no physical (anatomical) demonstrable facts can be established in reference to the subject.

2. *Diseases of the Texture of the Heart.*—To these belong:

a. First, and most prominently, *inflammations*, as for instance of the pericardium, the muscular substance of the heart and the endocardium, both in their primary and secondary character. By paralyzing the substance of the heart, inflammations occasion dilatations, which are maintained by their own secondary conditions, which mechanically augment them, and gradually superinduce hypertrophy.

An important place must be assigned to dilatations arising from chronic pericarditis, especially when associated with purulent exudation, or when investing the heart with a pseudo-membrane incapable of contraction, or lastly, when there is firm adhesion of the heart to the pericardium consequent on these new structures. Inflammation, in proportion to its intensity, and the quantity and purulent character of its effusion, tends to promote paralysis of the muscular substance of the heart, accompanied by decoloration and diminished cohesion, and hence furthers the development of passive dilatation. The longer the inflammation has continued, the more permanent will be the character of the cardiac affection, and if at length the heart adheres to the pericardium, a mechanical obstruction is opposed to the contraction of the former by the pseudo-membrane, which agglutinates the pericardium to the heart. This form of dilatation commonly affects the whole heart.

The dilatations induced in the same way by the endocarditic process, are similar to the former, but are usually less important; and when it gives rise to anomalies of the valves, they may gradually assume the active form by the association of hypertrophy. The left side of the heart, especially the left ventricle, is incomparably the most frequent seat of these affections.

It must be evident that the importance of the dilatation induced by the inflammation of the muscular tissue of the heart, will be in proportion to the frequency of inflammation, and the number and extent of its starting-points. This form of dilatation, excepting in very rare cases, invariably affects the left ventricle.

b. Adiposity of the Heart.—An excessive accumulation and formation of adipose tissue in the heart promotes dilatation of a passive character, in consequence of the simultaneous attenuation of the muscular walls of the heart.

The form which we regarded as consecutive, in speaking of the condition of the texture of the hypertrophied and dilated heart, appears to favor further dilatation.

3. Finally, in all those cases in which cardiac disease cannot be referred to any of the above-enumerated causes, it may originate in excessive *innervation* of the heart. Under this head we may include a considerable number of cases of hypertrophy and dilatation of the left side of the heart, which Bouillaud has termed *primary*, in order to distinguish them from consecutive forms arising from the causes already indicated.

Many of these causes, more especially endocarditis and its secondary conditions, are occasionally observed in the fœtus, and the diseases to which they give rise under these circumstances are then *congenital*. Other cardiac diseases belonging to this category depend on *original malformation* of the heart, its ostia and vascular trunks, and constitute a special series, of which we purpose treating subsequently under the head of cyanosis.

Independently of these causes, the *consecutive diseases, arising from affections of the heart*, are alike important and numerous. The following are the most worthy of notice:

Excessive Fulness and Dilatation. Stasis in the whole Venous System.—This condition is most strikingly manifested in the great venous trunks, the venæ cavæ, and the trunk of the portal vein,—from whence it extends along their branches into the capillaries, and is then characterized by distension and cyanosis.

Hemorrhages, resulting from the excessive fulness of the capillary system, manifested in discharges of blood from the mucous membrane of the nose, excessive menstrual uterine discharge, bleeding from the bronchial and pulmonary mucous membrane (hæmoptysis and hæmoptoic infarctus), hemorrhage from the intestinal mucous membrane, from the liver (apoplexy of the liver), and into the brain. The most frequent and important of these, are bleeding from the bronchial and pulmonary mucous membrane, and cerebral hemorrhage; and we will, therefore, treat of them specially, together with other subjects, in a future page.

Hypertrophies, more especially affecting the parenchymatous abdominal viscera, as the liver, spleen, and kidneys, although more particularly the two former. These affections are frequently marked by a visible increase in the volume of the organs, by a persistent tumor, and more commonly—either with or without the former,—by a striking increase of consistence depending on a compression of the elementary structure induced by hypertrophy.

To these we must add *hypertrophies of the mucous membranes and the chronic catarrhal inflammatory conditions—the forms of blennorrhœa*—to which they give rise. Those which are most remarkable for their intensity and extent are bronchial catarrh, and a catarrhal condition of the whole of the intestinal mucous membrane.

Dropsy which usually manifests itself first as anasarca of the lower extremities, and is then converted into general dropsy by the addition of serous effusions into the large serous sacs, is the result of the above-named venosity and mechanical hyperæmia. Œdema of the lungs is highly important, whether it be slowly established, and as a result of dropsy in other parts, or whether it show itself among the earliest symptoms of dropsy, and speedily attains a high degree of intensity, when it not unfrequently proves rapidly fatal.

Besides these secondary conditions, there are others which, from their importance, merit special consideration : these are certain *diseases of the liver*.

The diseases of the liver, of which we are about to speak, have frequently been regarded as causes of disease of the heart, but it is not very clear in what manner they can be supposed capable of bringing about such a result. Facts as well as theory tend rather to show, that the morbid condition of the liver is a consequence of heart-diseases, and is developed by the constant mechanical hyperæmia induced by the latter. To this class belong, besides the hypertrophy already described, the condition known as *nutmeg liver*, which is generally developed in a very intense form, that is to say, as a sharply-defined saturated yellow substance, very rich in blood, and marked with well-defined red patches; and, finally, that *granular condition of the liver* which is gradually developed from the latter, either with or without inflammation.

In our observations on hemorrhages, we spoke of *those of the bronchial and pulmonary mucous membranes*, and *of cerebral hemorrhage* as among the most important results of the cardiac diseases under consideration. Cerebral hemorrhage (apoplexia gravis) occurs in so large a majority of cases in conjunction with disease of the heart, that the latter has, with much reason, been regarded as a predisposing cause of cerebral apoplexy. The disease of the heart consists here in simple hypertrophy of the left ventricle, or what is much more frequently the case, in its dilatation, associated with highly developed hypertrophy. Cerebral apoplexy is undoubtedly induced by laceration of the cerebral vessels, occasioned by the augmented impulse propagated from the left ventricle; and this is the more easily effected the more the arterial coats in advanced life have lost their normal texture and cohesion, their power of resistance, and elasticity, or have become ossified, &c. A similar or even identical relation has been supposed to exist between hemorrhages of the bronchial and pulmonary mucous membrane and active dilatation of the right side of the heart. It must, however, be observed in reference to this point, that hemorrhages of this kind very frequently occur in dilatation and hypertrophies of the most different portions of the heart, in the form of hæmoptysis and hæmoptoic infarctus (pulmonary apoplexy). The cases in which they are found to exist, are very rare in comparison with the frequency of active dilatation of the right ventricle, and their coincidence bears a very secondary relation to the frequency of the coexistence of active dilatation of the left ventricle and cerebral apoplexy. Whether this depends on the absence of that diseased condition of the coats of the vessels in the branches of the pulmonary artery which is found to exist in the cerebral vessels, is a point that has not been determined, since, on the one hand, cerebral apoplexy, when associated with the above-named heart-disease (viz., hypertrophy of the left ventricle), is found to occur without any recognizable anomaly of the cerebral vessels; and on the other, because bronchial and pulmonary hemorrhage, are frequent in the cardiac diseases referred to. It follows, therefore, that these affections are only in very rare cases to be referred to an increased impulse propagated from the hypertrophied right ventricle, and that in the most numerous cases they are the result of an excessive fulness of the whole vascular apparatus of the lungs, induced by the obstruction opposed to the emptying of the pulmonary veins into the left side of the heart.

These diseases of the heart attack individuals of every age, not even excepting the fœtus, but they occur more frequently in advanced life than in childhood and adolescence, simply because the different causes favorable to their active development have been for a longer time in operation, and the system is no longer equally able to resist disease.

They frequently prove fatal in consequence of the secondary diseases to which they give rise, and often produce sudden death, especially by paralysis of the hypertrophied organ, hyperæmia of the lungs, rapidly developed pulmonary œdema, or cerebral hemorrhage.

They are further worthy of notice on account of the immunity from tuberculosis, which they insure to those affected by them; and it may be generally remarked, that the immunity which is yielded by the most various anomalies is always dependent on this class of diseases of the heart.

B. *Abnormal Smallness.*

Anomalous smallness of the heart appears under *two essentially different forms*, being either *congenital and original*, or the result of atrophy —*atrophied.* Abnormal smallness from either of these causes is of incomparably less frequent occurrence than excessive size.

The *former* of these conditions is occasionally associated with a fœtal conformation of the heart, patency of the foramen ovale, and even with more considerable malformations; but many cases present exceptions to this rule. The degree of abnormal smallness varies, the heart of the adult being in some well-marked cases no larger than that of a child of six or seven years of age. This condition appears to be most common in the female sex, and is not unfrequently connected with retarded development of the sexual organs, especially where this arrested development affects the whole system. The opinion expressed by Laennec, that frequent syncope is dependent on a heart too small in relation to the body of the individual, is worthy of observation.

Atrophy of the heart accompanies, to a certain extent, all general wastings of the body, being commonly observed after typhus, and especially in marasmus, in consequence of tuberculous and cancerous secondary formations and their disintegration. An atrophied condition of the heart is also occasionally produced by pressure and want of space, as for instance by bulky secondary products in the mediastinum, and is, moreover, also the result of pericarditis, accumulations of fat on the heart, &c. Contractions of the openings of the coronary arteries is an important and influential cause.

The heart itself differs under these circumstances, the tissue being either tough, and in that case usually of a reddish-brown color, or relaxed, easily torn, of a rusty fawn-color, and a faded appearance. According to Bouillaud, three different forms may be established in reference to the cavities of the heart, viz.:

a. Simple atrophy, wasting (attenuation) of the walls, with a normal condition of the capacity of the cavities.

b. Eccentric atrophy, attenuation of the walls with dilatation of the cavities.

c. Concentric atrophy, a normal or even an increased thickness of the walls, with contraction of the cavities; this is the most common form.

In the first form the volume of the heart is contracted; in the second it may be contracted, normal, or augmented; in the third it is constantly and generally strikingly contracted. This last form approaches most nearly to original smallness, with which it may even be confounded.

Besides the above named-characteristics of the muscular substance of the heart, other signs of atrophy may be mentioned, as, for instance, disappearance of the fat of the heart, serous infiltration of the adipose cellular tissue at the apex, the base, &c., in consequence of shrivelling of the opaque pericardium and of the milk-spots that may be present; and, lastly, an unusually winding course of the coronary artery.

Morbid attenuation, atrophy of the endocardium, and of the *valves*, will be considered in the sequel.

§ 5. *Anomalies of Consistence.*

We have already acquired some knowledge of several of these anomalies, to which belong:

An *increase of consistence* in the muscular substance of the heart in hypertrophies, which is occasionally very considerable, especially in the right ventricle;

A *diminution of consistence* in passive dilatations and in some forms of atrophy:

A peculiar *diminution of the consistence* of the muscular substance of the heart, associated with decoloration, and as the result of pericarditis, and more especially hemorrhagic, purulent, and tuberculous exudations, which impart to it something of the character of half-boiled meat. (See p. 111.)

Another form of *diminished consistence*, of which we have only spoken cursorily, and which will be considered more fully in a future page, is that which accompanies adiposity of the heart.

The cases that have been regarded by many observers as *softening of the heart's substance*, most probably belong to one or other of these forms of extreme diminution of consistence. It is not unlikely that many of these may have originated in an inflammatory centre in the tissue of the heart.

The diminution of consistence, or a relaxed condition in which the tissues can be easily torn, and which is occasionally observed as the result of typhus, is a mere symptomatic and simple diminution of consistence not depending upon any disturbance of texture.

Softening of the valves will be considered when we speak of the diseases of these structures.

§ 6. *Separations of Continuity.*

To this class belong:

a. Wounds of the heart produced by sharp thrusting instruments, as well as by the penetration of fragments of the ribs, sternum, &c. Such wounds, whether superficial or sufficiently deep to penetrate into the cavities, may injure the heart from different directions and at one or more points.

b. Ruptures of the heart induced by violent shocks implicate, according to circumstances, different portions of the heart, very frequently to a great extent.

c. Spontaneous Ruptures or lacerations of the heart. (*Cardiorhexis, Ruptura cordis spontanea.*) These are the most important of this class of lesions.

Such spontaneous ruptures affect either the walls of a cavity of the heart, a papillary muscle, or its tendinous portion only, a trabecula carnea, or a valve,—the first of these being, however, by far the most common. The left ventricle is the most frequently lacerated, the right only comparatively rarely, and the auricles most rarely of all. When there is laceration of the left ventricle, the lesion almost invariably affects the convex or anterior wall, and generally its middle portion near the septum, the cases being very rare in which the plane or posterior wall is lacerated. The laceration may generally be observed on the external surface of the heart in the form of a fissure, varying in length, and inclining inwards in the direction of the septum. This lesion presents a different appearance in the interior, for here we observe that the muscular substance is, as it were, bruised and crushed over a large extent, near the inner surface, the rent exhibiting either a straight or an oblique course, or in some cases a deep ramified cleft. A coagulum is very commonly found interspersed in the interior among the trabeculæ, or it occasionally fills up the whole cavity.

There is usually only one rent present, but there are occasionally two or even more rents, which are either wholly separate or connected under the surface, and appear at different distances from one another, even in wholly different compartments, as, for instance, simultaneously in the left and the right ventricles.

The investigation of the causes that give rise to these lacerations is a subject of great importance. However much we might be disposed to believe that a heart having thin, relaxed walls, which can be easily torn, would be pre-eminently liable to rupture, or that this lesion would be more common in those portions of the heart in which the walls are thinnest, such is by no means the case, for laceration takes place, as we have already seen, precisely in the thickest and strongest portions of the heart, and is usually found to occur where the organ is in a hypertrophied condition. On submitting such cases to a more careful investigation, we find, however, as has been already in part shown, that the portion referred to, viz., the left ventricle, in consequence of the diseased condition in which it is often found, especially when combined with hypertrophy, may be so intensely predisposed to spontaneous laceration that this lesion of the heart may occur during a condition of complete bodily and mental repose, and not merely under circumstances of increased action from various exciting causes.

Among the morbid conditions predisposing to rupture, we must place the various fatty conditions of the heart, more especially those which we have mentioned under the head of hypertrophies and dilatations, as a frequent form of textural lesion, in which the muscular substance of the heart assumes a dirty yellowish discoloration, may be easily torn, and becomes loose and flabby. Laceration of the heart is also frequently occasioned by centres of inflammation in the earlier stages, seated in the muscular substance. Contractions of the opening of the aorta may also be included among the remote causes, whilst advanced age affords a specially predisposing cause.

These lacerations, as well as penetrating heart-wounds, generally terminate speedily in death. It has been asked where we are to seek the cause of the speedy occurrence of death in those cases where the quantity of blood extravasated in the cavity of the pericardium is not sufficient to account for the fatal termination of the disease. Some have referred the cause to the implication of the function of the heart itself, in consequence of the extravasation and of the separation of numerous muscular fibres in extensive wounds of this organ. Besides these causes, Bouillaud has advanced an opinion deserving of attention, that death results from syncope,—anæmia of the brain—owing to the sudden abstraction of blood induced by extravasation from the left ventricle.

Penetrating heart-wounds are not, however, invariably rapidly fatal; life being in some cases considerably prolonged, while it would even appear, in accordance with some observations selected from a large number of cases, that wounds of the heart may be occasionally followed by recovery. The fact that death does not immediately ensue has been explained on the ground of the narrow and oblique course of the wound, and by the different position and crossing of the various wounded layers of the muscular substance. In many cases the wound has been closed by some portion or the whole of the instrument, or even a fragment of a rib, remaining imbedded in it.

It is difficult to answer the question whether the rent occurs in spontaneous ruptures, during the systole or the diastole. Many (Pigeaux, amongst others) are of opinion, although without sufficient ground, that it generally takes place during the diastole. Judging by analogy with lacerations of the voluntary muscles, it must take place during the systole. In proof, however, of the frequency of its occurrence during the diastole, the fact might be advanced that the course of the rent in the heart, when contracting after the hæmorrhage, is not straight, but angular or zigzag, in consequence of the disturbed position of the different muscular layers.

It is equally difficult to determine, generally or in any individual case, whether the rent has affected the whole thickness of the heart at once, or whether it has proceeded gradually, till it finally penetrated the whole thickness of the wall, and whether it began at the exterior or in the interior. According to our observations on this subject, the rent begins, in most cases, in the inner muscular layers. The laceration of a papillary muscle, or of a trabecula carnea, is of very rare occurrence, and the conditions on which it depends are probably the same as those of fissure of the heart's wall.

Laceration of the tendons of the papillary muscles and of the valves probably always depends on the relaxation and lacerability of the tissue, associated with inflammation of the lining membrane of the heart (endocarditis). It not unfrequently acquires importance by the valvular insufficiency to which it gives rise. Læsiones continui of the valves will be more fully considered in a future page.

§ 7. *Diseases of Texture.*

a. Hyperæmia, Anæmia.—We are not acquainted with any special condition characteristic of *hyperæmia of the heart.* Occasionally however, hyperæmia, as it manifests itself in the hypertrophies and dilatations which arise especially from stenosis, and in asphyxia in new-born infants and adults, is marked by the dark color of the muscular substance of the heart, and by a fulness of the vessels, more particularly of the veins, and in its more highly developed stages, by slight extravasation in the form of ecchymoses, about the size of millet-seeds or lentils, especially in the external strata and near the base of the heart, at the auricles, and in the vicinity of the origin of the arterial trunks.

Apoplexy of the Heart, manifested by an extravasation of blood into the muscular substance,—a suffusion of the muscular tissue,—is a symptom of no importance in the various degrees of laceration of the heart.

Anæmia of the Heart is probably often overlooked on account of the indistinct signs by which it is characterized. Such a state constitutes, however, a very important (but as it would appear, a hitherto disregarded) morbid condition, as we may learn from the contractions and final obliterations of the openings of the coronary arteries occurring in diseases of the aorta.

b. Inflammations.—After having spoken of inflammation of the external investment of the heart—pericarditis,—it still remains for us to notice *inflammation of the lining membrane,* and *of the muscular substance of the heart.*

1. *Inflammation of the Lining Membrane of the Heart, Endocarditis.*—It is only in modern times, and from the observations of Bouilland, that this species of inflammation, under the name of endocarditis, has been shown to be the special basis of numerous consecutive heart-diseases. The importance of the subject, both intrinsically and with reference to the different opinions advanced regarding the frequency of the disease, the absence of any well-founded data for its correct diagnosis after death (notwithstanding the many attempts made for their establishment), and, lastly, our still inaccurate knowledge of its course, its termination, and sequelæ, &c., have determined us to precede our general notice by a few explanatory observations; and at the same time we would simply remark, as will be seen in this section, that we have arrived, with reference to some points, at a totally different conclusion from the opinions usually expressed regarding endocarditis.

The endocardium corresponds with the inner coat of the vessels, and consists essentially, besides the epithelium, of a longitudinal fibrous coat (Henle), under which there is a very considerable layer of elastic and cellular tissue, which is most distinct in the auricles, and especially in their atria, and on which rests the muscular substance of the heart. In the left side of the heart, more especially in the left auricle, a layer similar to that of the circular fibres of the arteries is occasionally found under the longitudinal fibrous coat. This compound investment covers the trabeculæ carneæ, the papillary muscles, and their tendons, while the true endocardium invests the valves also, which, however, can only be regarded as duplications of that membrane, if we consider them as essentially composed of a fibrous tissue supplied with vessels. Besides this fibrous tissue, which is composed of a cellular-fibre-like substance, and delicate nucleated fibres, we also find unstriped muscular fibres in the auriculo-ventricular valves in individuals having a robust and muscular frame. The internal layers of this integument (the epithelium and longitudinal fibrous coat, which constitute the true endocardium) are devoid of vessels; but such is by no means the case with respect to the subjacent cellular tissue, which is permeated with numerous elastic fibres, or with the muscular substance of the heart. The endocardium, as we find in dilatations of the cavities of the heart, and in enlargements of the valves, arising from dilatations of the ostia, is capable of undergoing considerable expansion and attenuation. It is much thicker in the left side, and especially in the left auricle, than in the right side of the heart.

The relation of the true endocardium (the epithelium and the longitudinal fibrous coat) to the subjacent layer furnished with vessels, corresponds with that existing between the inner coat of the veins and their external coats. This condition affords *à priori* evidence of the possibility of inflammation of both coats, considering it, in its usual sense, with exudation of the free surface, whilst there is no inflammation, properly so called, in the inner coat of the arteries, at least not in the larger vessels, having a thick, yellow, muscular coat of circular fibres. The actual seat of these inflammations is the cellular substance lying under the endocardium and the inner coat of the vessel; we must, therefore, suppose, that in cases where products of inflammation are deposited on the inner surface of the heart or of the vein, the exudation must have penetrated through the permeable texture of the endocardium or through the inner coat of the vessel, or that the latter has been removed, either by solution or fusion, by means of the process of exudation. The latter condition will naturally be found to be of most common occurrence in inflammations having a purulent ichorous exudation. Such alterations manifest themselves by opacity, lacerability, and a felt-like porosity of the endocardium, excoriation of the subjacent layers, &c.

We learn from the foregoing observations how far the designation *endocarditis* is applicable to inflammation of the lining membrane of the heart. Thus, for instance, it is evident that since the endocardium, like the inner coat of the vessels, is non-vascular, it cannot be the seat of inflammation, which affects merely the tissue lying immediately below it, which is furnished with vessels. We purpose retaining the term endocarditis in this sense, discarding its use in reference to the valves, for which we shall simply retain the designation of inflammation of the valves.

Although endocarditis is a disease of very frequent occurrence, it must not be supposed that the term is applicable to all the diseases ascribed to it, its products, and sequelæ, for, as we shall soon learn, many morbid conditions of the valves of the left side of the heart, especially of those of the aorta, are the products of the same process which manifests itself in the arteries as a morbid deposit on the inner coat.

Endocarditis attacks different portions of the lining membrane of the heart, affecting in some cases the endocardium covering the inner surface of a cavity, the papillary muscles, and the trabeculæ, in others that of the valves, while in others again it affects both. Endocarditis of the valves is the most frequent and the most important, from the consecutive heart-diseases to which it gives rise. We will consider the signs of both under one head, referring specially to the peculiar characters of endocarditis of the valves.

1. *Redness and injection.*—In order that these conditions may be regarded as the manifestation of inflammation, it is necessary that the former should be the result of the latter (inflammatory injection or stasis), or that it should depend on an exudation containing hæmatin into the tissue. This latter form of redness is always found together with other signs of inflammation, and usually presents a mottled appearance. Where the redness cannot be referred, at least in part, to the above cause, it cannot be regarded as a sign of inflammation. Now, in point of fact, all the different forms of redness of the endocardium, which have been generally described as characteristic of endocarditis, belong to the latter class, and the descriptions given of these various forms evidently show that they are mere modifications of that redness which depends on infiltration of the tissue with hæmatin. There are, however, so few opportunities of detecting the peculiar redness of the endocardium arising from an injection, as our own numerous observations can testify, that it would not be surprising if anatomists, instead of committing an error of this nature, had wholly denied the existence of inflammatory redness in endocarditis. It is only in the first stage of the disease that a true redness and injection can be observed through the endocardium; it is only, therefore, in the very rare cases in which death occurs in the earliest stage, either from this or some other disease, that this condition of redness can be perceived. In most cases a redness from imbibition, resulting from the diseased condition of the blood, is *actually* present, and renders it extremely difficult and almost impossible to discover the redness from injection, which differs wholly from the above-named red colorations, and is constantly of a pale, rose-red color, whose tint is subdued by the endocardium covering it. Its appearance is never that of a saturation of the tissue, and its stripe-like, ramifying course, corresponding to that of the vessels, may the more easily escape detection, when it is concealed by the presence of a simultaneous red coloration, arising from infiltration. In most cases we are unable to perceive this redness from the circumstance of its being wholly masked by the conditions we are about to consider, viz.:

2. *Opacity and thickening of the Endocardium.*—In consequence of the extension of the process, the endocardium at various differently-sized spots is rendered opaque, whitish, and milky, whilst at the same time it becomes more or less thickened and swollen. This opacity and thickening depend on the deposition of the product of inflammation in the

tissue of the endocardium and the subjacent stratum, where it either solidifies or exerts a relaxing, macerating, solvent action on that tissue. The opaque and thickened parts are not clearly defined, but appear gradually to lose themselves in the adjacent portions of the endocardium. Valves affected by endocarditis exhibit a remarkable degree of thickening, because the substratum of infiltration—the tissue occurring between the two lamellæ of the endocardium—is here accumulated in large quantity. The shining smooth appearance of the endocardium vanishes with the increase of the opacity and thickening, and it then acquires a *dull*, velvet or felt-like and *rough* surface.

3. The whole of the lining membrane of the heart acquires *a looseness of texture*, and then readily admits of being torn, while the true endocardium is easily detached. In inflammation of the valves, their fibrous tissue very frequently appears to be in an extreme state of looseness and relaxation.

4. *Products of Inflammation.*—To this class belongs the above-named infiltration of the endocardium and of the subjacent tissue, but the question here arises, whether there is also exudation on the free surface of the endocardium, and how far such a condition is necessary to establish the existence of endocarditis.

The fact of such an exudation being deposited on the free surface of the endocardium in most cases of endocarditis, is rendered highly probable, not only from the results of pathological investigations, and the analogy presented by inflammations of other similar structures, especially the serous membranes, but still more so from the symptoms manifested during life. This exudation at the moment of its production merges into, and is taken up by the mass of the blood, where, in accordance with its character and intensity, it gives rise to the different general symptoms manifested during life, and to the characteristic secondary processes observed in the capillary system in endocarditis. In many cases, however, this exudation, doubtless in consequence of a very high degree of coagulability, remains on the inner surface of the endocardium in the form of a membranous coagulum, having a delicate felt-like, or shaggy free surface, which we have rarely an opportunity of seeing in its original condition, but which may very frequently be subsequently observed under different forms, but most distinctly in the form of milk-spots on the endocardium. In endocarditis of the valves it commonly manifests itself in the form of felt-like or granular masses, under which the valve appears rough, loose in its texture, and excoriated, and it then, in part, constitutes the so-called vegetations of the valves of the heart. We shall subsequently speak of purulent exudation on the endocardium.

5. *The so-called Vegetations or Fibrinous Coagula* which occur under the most various forms, more especially when they appear on the valves of the heart, are generally, and without exception, regarded as characteristics of endocarditis. As, however, they are not invariably direct products of an exudative process, but, on the contrary, in some cases wholly, but more frequently only in part, indirect effects of endocarditis, since they undoubtedly also appear independently of that disease, we cannot regard them as signs of endocarditis without some limitation in

accordance with what we have already stated, and with that which we purpose advancing in a subsequent part of this work, when we proceed to treat specially of vegetations in the heart.

It follows from the above considerations that the anatomical characteristics of endocarditis are very inconsiderable in number, when compared with those of other inflammatory affections; redness and injection are only seldom to be observed, an inflammatory product on the free surface of the endocardium is not always to be detected, and the vegetations are only conditionally a sign of endocarditis. There remain, therefore, as the only constant signs, opacity and thickening of the endocardium, with the disappearance of the smoothness and polish of its surface. But as these conditions of opacity and thickening of the endocardium may, as we have already remarked, be produced by a process wholly different from that of endocarditis, it will be readily understood how difficult is its diagnosis, and how easily its products may be confounded with those of some wholly different process.

In the above delineation we have purposely limited ourselves to the most important points, in order to give a general sketch of the endocarditic process; and with the further view of not disturbing our readers by any superficial details, we have described only the characters presented in the most numerous and common cases of endocarditis. We purpose considering this subject with the completeness which its importance demands, and we will then treat of all those points that have been neglected in the present portion of our work. The following observations will contain a notice of many of the more uncommon events occurring in the course of endocarditis, and of many appearances and processes which have merely been briefly indicated in the preceding delineation, together with the terminations, sequelæ, &c., of the disease.

a. In intense forms of endocarditis, *a separation of continuity* of the structure affected by the inflammation not unfrequently manifests itself as a highly important occurrence. It may occur in different ways, either as laceration of a valve, or of one or more of the tendons of the papillary muscles, or of the endocardium on the wall of the heart. This separation of continuity is the final result of a maximum degree of inflammatory loosening of the tissue. The margins of the fissure are generally jagged, and serve as the places of deposit for a large number of vegetations. The tissue of the torn structure, as for instance of a tendon, is usually considerably reddened, infiltrated by inflammatory products, and easily torn. Laceration at the wall may give rise to the formation of aneurism of the heart, whilst, if it affect the valve, it may, under certain circumstances, occasion valvular aneurism.

b. Endocarditis with purulent exudation is not of very uncommon occurrence; and although the recognition of the seat and position of pure pus, as a free product, is, in most cases, impracticable, it is not difficult to prove the extreme probability of the existence of such a process. The loosening of the tissue, the want of polish, and the felt-like character of the endocardium, are very strongly marked in the centre of inflammation, and hence these lacerations frequently occur. In these cases a purulent product mixed with blood is generally found infiltrated into the tissue, if not at the surface of the endocardium, whilst abscesses are occasionally found to have spread themselves over a various extent of surface below the endocardium, in the cellular and adjoining muscular strata, deep in the tendons, and in the tissue of the valves. Finally, the process of suppuration being established, an ulcerous separation of continuity will be effected in various ways, in the endocardium of the walls of the heart, in a tendon, or in a valve. The vegetations deposited on the ulcerated surface and its margins are remarkable for their excessive number, their inconsiderable consistence, bad color, and their tendency to purulent disintegration. The secondary processes in the capillary system terminate in purulent solution, whilst the intensity and malignant character of the general symptoms during life lead us to conjecture that some deleterious substance has been taken up into the blood.

c. *Endocarditis is probably always an acute disease;* it may, however, frequently recur, and at the same spots; but we cannot admit the existence of a chronic form of the disease, unless, according to Bouillaud's incorrect view, we regard as such the symptoms manifested during life by its products, and the further development and metamorphosis of those products, that is to say, the terminations and sequelæ of endocarditis as given below.

1. *Exudations on the free surface of the Endocardium* in the form of agglutinated, whitish, or bluish-white laminæ of different size and form, resembling in appearance a serous or fibro-serous membrane, under which the endocardium appears normal, or scarcely at all opaque. They at one time appear in the form of narrow stripes, at another in that of more considerable, irregular plaques or patches, varying from the size of a silver groschen to that of a zwanzigerstück [a coin rather larger than a shilling], and admitting of being easily removed from the endocardium, over which they are in general smoothly drawn or occasionally compressed together in folds. They are most frequently observed in the left side of the heart, at the upper part of the septum towards the aortic opening, where they are puckered and drawn aside into plaits by the blood flowing over them. Their texture resembles that of the longitudinal fibrous coat, and they consist in some cases of thick stiff fibres, and in others of soft fibres of areolar tissue. The facility with which the agglutination of the inner milk-spots are severed, and the laceration of texture occasioned by their separation, cause them to differ very widely from other structures. The milk-spots are, however, almost always sharply defined in these cases.

2. *Permanent Thickening of the Endocardium and of the Subjacent Tissue* becomes the more considerable in proportion to the intensity of the endocarditis, and the frequency of its occurrence at the same spot. It is generally occasioned either by infiltration into the tissue, or by exudation that has solidified and become organized on the free surface of the endocardium; the former of these exerts, however, a preponderating influence, as is especially observed in the valves. Thickening is manifested in the walls of the heart in the form of patches of various extent, in some of the trabeculæ as a tendinous ring or sheath, in the papillary muscles as a tendinous covering over their extremities, in the tendons themselves as a wad-like or spindle-shaped thickening, and in

the valves as a more or less uniform thickening of their free margins, extending from thence to various distances, and even across the valve towards their margin of insertion. The diseased tissue appears opaque, thick, tough, and of a white color, inclining to yellow; and it is with difficulty that the free exudation and the tissue infiltrated by solidified products of inflammation, which constitute the principal elements of the morbid mass, can be torn or split asunder, both having coalesced, and presenting a single fibroid and compressed texture.

The thickening of the tissue of the wall of the heart is often made more apparent in endocarditis by the association of inflammation of the contiguous stratum of muscle to various depths, which gradually passes into induration, and leaves a fibroid callus in the place of the muscular fibres.

3. *Coalescence* is frequently associated with this thickening of the tissue. As the thickened tissues coalesce with the free exudation, so also the latter may occasion a fusion of various tissues. In this manner the trabeculæ enclosed in tendinous sheaths unite with one another or with the walls of the heart, while the same process may be observed amongst the separate points of a papillary muscle, or the tendons of a papillary muscle may merge into either one or several strings, or the different valves may coalesce with one another, or with the wall of the heart or of the vessel.

4. This fibroid mass of exudation exhibits here, as in other places, a marked tendency to shrivel, by which means a *shrivelling or shortening* of the thickened structure takes place. To this class belong shortening of the papillary tendons, and a shrivelling of the valves associated with various malformations. The wall of the heart is either very indistinctly or not at all shrivelled, since it is raised by the substratum of muscle in those cases where the latter has retained its normal texture and function; the adventitious product is expanded rather than shrunk, owing to the great influx of blood in those cases in which the muscular substance of the heart has been reduced to a state of paralysis by the action of inflammation, or has suffered a change of texture.

5. *Calcareous Concretions* become developed sooner or later in the fibroid secondary product, and appear in rare cases in the form of nodular uneven laminæ in the thickened endocardium of the wall of the heart, and more frequently as simple nodular or ramified strings or rows, or even as amorphous masses of various thickness in the tissue of the thickened valves, and of the thickened papillary tendons which are generally fused and blended together.

6. We have already fully considered the subject of endocarditis terminating in *Suppuration*.

d. Although endocarditis is generally characterized by the terminations and sequelæ already indicated, the cases in which it terminates by a perfect cure are not of very rare occurrence, as we learn from careful observations on the living subject, and by a correct interpretation of the appearances presented after death. This favorable termination depends occasionally on a complete resolution; or, in other words, on the absorption of the products of inflammation deposited in the tissue, and on the fact that the portion of the free exudation which is solidified on the endocardium, and the vegetations that may be present, are gradually taken up and mixed with the mass of the blood in the form of finely-divided molecules. In some cases fragments which, from their size and position, do not constitute an impediment to the circulation, may remain; or, again, in other cases partial thickening of the valves is counteracted and rendered inoccuous by their becoming attenuated at one or more points, by a shortening of one or more of the papillary tendons, or an elongation of the muscle or of the extremity of the diseased valve.

e. The Vegetations on the Valves above referred to undergo different metamorphoses, as we have already seen, and as will be made more apparent in the sequel. We will here specially notice:

1. *Their gradual Diminution and Final Disappearance.*—According to numerous highly interesting analogies, it would appear that these conditions depend on an actual waste of the fibrinous coagulum. They undoubtedly occur very commonly, and from a comparison of the frequent or almost invariable appearance of very numerous and extensive vegetations on the valves in recent endocarditis, and their insignificant character and occasional absence in obsolete cases, it appears evident that in the course of time, they become considerably diminished, and at length entirely disappear.

2. *Ossification and Calcification* of these vegetations are metamorphoses, which, although of frequent occurrence, have not hitherto been duly considered. They constitute a special form of valvular ossification, which has never yet been duly considered.

f. The secondary Coagulation of Blood in the capillary system, together with its metamorphosis, which presents a highly important indication of the endocarditic process,[1] has also been disregarded by observers. It indicates the most important phenomenon manifested during endocarditis, namely, the formation of a product on the free surface of the endocardium and its absorption into the mass of the blood, and consequently shows the equal importance of endocarditis and inflammation of the vessels (namely, of the veins), while it moreover tends to elucidate the symptoms of disease during life. It is more constant than in phlebitis, inasmuch as, from the absorption of the inflammatory product, no coagulum can be formed in the heart of a similar nature to those which occur in the veins, and hence there can be no immunity afforded against a poisoning of the whole mass of the blood. This process probably, on this account, constitutes an important means of diagnosing between obsolete endocarditis and a form of hypertrophy of the endocardium, and more especially of the valves, which is induced by depositions from the blood; but this subject we will presently consider more at large.

It is of common occurrence in the spleen and kidneys, but is seldom found in the lungs, excepting in the very rare cases of endocarditis of the right side of the heart. The secondary processes which result from endocarditis depositing a purulent exudation, and terminating in suppuration, are less limited to these organs of hæmatosis, and manifest themselves as metastases in the subcutaneous cellular tissue, in the mucous membranes, &c.

The process commonly called Phlebitis (but more appropriately termed

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[1] Oesterr. med. Jahrbücher, B. xix. St. 3.

Angioitis capillaris) consists, as far as we know, like that observed in a larger vessel (namely, a vein), in a coagulation of the blood in the capillaries, and a metamorphosis of the coagulated fibrin, varying in accordance with the quality of the absorbed product. Since endocarditis, in ordinary cases, yields no deleterious product (pus or ichor), the metamorphosis consists in a conversion of the fibrinous coagulum into a fibroid mass, with obliteration of the vessels, and so great a degree of obsolescence of the affected tissue of the diseased organ, that the whole resembles a cellulo-fibrous callus which shrivels to a callous, whitish or black cicatrix, containing pigment. It is not improbable that the process may terminate in resolution, or, in other words, in the solution of the coagulum, and thus leave no trace of its existence. In the very rare cases in which endocarditis deposits a purulent product, the coagulum in the capillaries becomes decomposed into a fluid, which is more or less purulent, according to its elementary composition, while there is consecutive fusion of the walls of the vessels and the diseased tissue, the result of which is the formation of an abscess, or so-called purulent metastasis. This form of endocarditis may result in a true process of exudation in the serous and synovial membranes, and even in the parenchyma, in consequence of the diseased condition of th blood induced by the morbid product.

g. Endocarditis by its proximate, no less than its secondary results, and therefore by a twofold local cause, may give rise to *Dilatations of the Heart*. As we have already observed in treating of these diseases, the form of dilatation thus occasioned is of a passive character, and depends on paralysis of the muscular substance of the heart, which is implicated in the inflammation. The dilatation is moreover mechanically increased in the more remote sequelæ of extensive endocarditis by the continuance of a morbid condition of the valves, which are almost invariably implicated; and in these cases a moderate degree of hypertrophy is gradually associated with the dilatation. The dilatation must also, as is evident, be more considerable from its very origin, and must be of a more decided passive character, where endocarditis has been combined with pericarditis and carditis; and where the latter affection is of a very intense and deep-seated character, endocarditis may give rise to true aneurism of the heart.

Endocarditis occurs with a preponderating degree of frequency in the left side of the heart, where it is also generally present in the very rare cases in which it attacks the right side. In the case of the former, both the ventricle and the auricle are affected, while in the case of the right side of the heart, the ventricle is the special seat of the disease. The auriculo-ventricular valves of the left side are more frequently diseased than those of the right, whilst many morbid conditions of the aortic valves cannot be actually referred to an endocarditic origin.

An interesting exception to these relations is presented in the fœtus, where endocarditis is much more frequent in the right side; and many of the cases of contraction of the openings of the right side, which are met with in childhood and youth, are undoubtedly congenital and of fœtal origin. There are, moreover, many anomalies of the arterial opening, especially of the right side of the heart and of its valves, which are commonly regarded as malformations (as, for instance, contraction and occlusion of this opening, and an abnormal condition of the trunk of the pulmonary artery), which are most probably the results of endocarditis already existing in an early period of fœtal life, and which give rise to many arrests of structure within the heart. We may undoubtedly explain in a similar manner the many endocarditic metamorphoses observed in the hearts of persons suffering from cyanosis.

From what has been already stated, it will appear, that endocarditis occurs in the fœtal condition as well as after birth. Youth and adolescence are the periods in which this affection is most frequently manifested.

The most important diseases with which it is associated, are its primary combination with pericarditis, and, whether this be present or not, with inflammations of serous membranes, namely, those of the synovial membranes—rheumatic inflammations of the joints. Valvular endocarditis, implicating tendinous insertion of the mitral valve, when combined with pericarditis, is extremely important, owing to the peculiar formation of the consecutive metamorphoses. Thus, for instance, we observe that the calcareous band developed at the tumefied point of insertion, not unfrequently expands into an osseous mass seated in the pseudo-membranous agglutinating medium between the pericardium and the heart. Endocarditis is also occasionally combined with carditis—inflammation of the muscular substance,—and this combination is then the common occasion inducing aneurism of the heart; while in some cases endocarditis may be merely an incidental combination, arising from some centre of inflammation in the muscular substance adjoining the endocardium.

To this class belong the combinations with croupous pneumonia, acute inflammation of the periosteum, acute ostitis, &c.

Endocarditis and its sequelæ are not unfrequently met with in combination with Bright's disease, which is probably to be explained by the fact that this heart-disease becomes associated with disease of the kidneys in consequence of the abnormal condition of the blood.

Many of the anomalies already partially considered occur as the remote and indirect sequelæ of endocarditis. Fœtal endocarditis, at an early period, obstructs the completion of the inner structure of the heart, by means of the results to which it gives rise, and especially by contracting the openings of the heart; when it occurs at a later period and after birth, it obstructs the involution (closure) of the fœtal passages. In subsequent periods of extra-uterine existence, many of the diseases of the different systems and organs considered under the head of dilatation and hypertrophy, may still be traced to the dilatations of the cavities of the heart and the anomalies of the valves (contraction and insufficiency), which have their origin in fœtal endocarditis.

Hypertrophy and Atrophy of the Endocardium.—By these conditions we purpose indicating a thickening of the true endocardium (which, in respect to the main character of its composition, corresponds to the inner coat of the vessels), by a morbid deposition from the blood of a substance which becomes metamorphosed into the layers of the epithelium and longitudinal fibres composing the endocardium. This excessive deposition of new layers of the endocardium is a process which occurs in its most fully developed form in the arteries, and more especially in their main trunks, and will be duly considered in the appropriate place. Its proximate result is a thickening of the endocardium.

This morbid condition acquires additional importance from the facility with which it may be, and no doubt very frequently is, confounded with endocarditis and its products, which it greatly resembles, and with which it is often found associated. We have, on this account, thought it best to devote the closing part of the present section on endocarditis, to the consideration of this subject, however unusual such an order of arrangement may appear. (See p. 123.) The following remarks on the peculiar characteristics of this affection will clearly exhibit the differences which distinguish it from the endocarditic process and its products.

In the lower degrees it is only by a careful investigation that we can discover any undue thickening of the endocardium. The color of the muscular substance is less clearly discernible, while more strata than usual must be removed before we reach the layer of cellular tissues interspersed with elastic fibres, which is situated under the endocardium; moreover we clearly observe that the innermost layers are lighter and softer, and that the tissue which constitutes the longitudinal fibrous coat is less developed and more moist.

In this manner new depositions of layers of endocardium, either with or without an epithelial investment, are frequently found to cover one or more of the cavities of the heart (the ventricle or auricle of the left or both sides), together with the corresponding valves.

When this process of deposition has been frequently repeated, and the thickening of the lining membrane of the heart is correspondingly increased, this condition will be easily recognized. In these higher degrees of intensity we very frequently observe, as in the arteries, that the endocardium exhibits, at more or less well-defined spots, portions of thicker surface in the form of islands or patches, while we at the same time remark that the valves, more especially those of the aorta, have been considerably thickened and enlarged by the deposit.

The opalescent translucence and stratification of the deposit and the uniform texture of the combined lamellæ, distinguish it from the products of endocarditis, from the loosely-attached, bluish-white, opaque milk-spots, and from the fibroid thickening of the endocardium, which cannot, without extreme difficulty and effort, be separated into strata, and which exhibits greater density and dryness of its tissue, and evidently consists of fibrous or areolar tissue. The absence of redness and injection in every stage and of vegetations and secondary processes (metastases) in the capillary system, distinguishes it from the endocarditic process. The existence of the process of deposition in the trunk of the aorta affords us further diagnostic aid in determining hypertrophy of the endocardium.

A correct diagnosis, which has for its object to determine both processes generally, and to distinguish the special share taken by each in the anomalies under consideration, is rendered more difficult in those cases in which, as we have already observed, the products of endocarditis occur simultaneously with the condition we term "excess of endocardiac formation," and which is indeed very commonly favored or even occasioned by the residua of endocarditis. A peculiar difficulty presents itself, when the deeper or older deposits lose their transparency, and become completely opaque, white or faded, in consequence of an atheromatous process, or of a metamorphosis tending to ossification. They may be distinguished from endocarditic products on a closer inspection, by the occurrence of a large quantity of molecules, consisting of albumen, fat, and calcareous salts, deposited in the different strata.

This metamorphosis never, so far as we know, proceeds on the walls of the heart, beyond the incipient stage above indicated; we have never found it developed in the true atheromatous process, nor have we ever been able to ascertain that this process formed the basis of any of the numerous cases of aneurism of the heart which we have examined. The valves, however, occasionally present the appearance of an incipient atheromatous disintegration of the deposit, while ossification of the deposit on the valves, more especially on those of the aorta, is very frequently a final result. It is highly probable, moreover, that all the forms of ossification of the valves which become developed in the advanced periods of life, belong to this class.

Hypertrophy of the endocardium is limited almost exclusively to the left side of the heart, and of the two arterial trunks it only attacks the aorta. The aortic valves and the left ventricle are more frequently and more intensely affected than the auriculo-ventricular valves and the auricle. The aorta is at the same time diseased in like manner, but generally in a very preponderating degree. The endocardium of the left auricle is, however, excessively thickened in some few cases where there is contraction of the mitral valve.

Endocarditic hypertrophy, like that of the aorta and its ramifications, especially occurs in advanced periods of life, and undoubtedly constitutes the source from whence arise a great number of those diseases of the aortic valves,—as, for instance, thickening, shrivelling, ossification, and insufficiency,—which are slowly developed in maturity and old age, without the pre-existence of endocarditis. This fact presents many points of great interest, when considered in relation to diseases of the mitral valves, which usually occur in young persons as a result of well-marked endocarditis.

This affection is frequently occasioned and favored by pre-existing dilatation of the heart and contraction of the openings, in consequence of which the blood is detained in the different cavities, and its further circulation impeded.

Atrophy or Attenuation of the Lining Membrane of the Heart is very seldom sufficiently manifested to come under notice. This membrane is certainly found to be uncommonly thin and transparent in some cases of dilatation of the heart; while we have remarked the same appearance in excessively fat hearts.

2. *Inflammations of the Muscular Substance of the Heart, Carditis (in the strict sense of the word), Myocarditis.*—Although inflammation of the *Muscular Substance of the Heart* is less frequent than endocarditis, it is much more frequent than is usually supposed. Its anatomical characters and its terminations are the same as those exhibited in inflammation of the muscular substance generally, but there are, nevertheless,

many points connected with this subject which demand special notice, both on account of their importance and peculiarity.

It occurs independently in the middle layers of the muscular substance most remote from the pericardium on the one hand and from the endocardium on the other, and in original or consecutive combination with pericarditis and endocarditis. The pericardium and the endocardium are always implicated in inflammation of the adjacent layer of muscle and conversely intense pericarditis, and more especially intense endocarditis influence the adjacent structure to various depths. It moreover most frequently affects the true fleshy walls of the heart, but sometimes its trabeculæ, and in some cases both simultaneously.

It also commonly occurs in the form of larger or smaller centres which are in some cases spread over a large portion of one cavity of the heart (as, for instance, the left ventricle), in which case, the wall of the heart is found to be affected throughout more or less of its thickness, when the disease is associated either with pericarditis or endocarditis singly or with both conjointly. In some rare cases one portion of the heart is found to be so thoroughly affected, that there are only a few layers of the muscular wall which are not implicated.

The seat of the affection is almost exclusively the left ventricle, which it attacks at every point, although less frequently at the septum; the apex is commonly attacked when the disease is very extensive. The right ventricle is very rarely affected, although we have observed the disease in an intense degree of development in the anterior wall of the conus arteriosus. It is of very rare occurrence, as far as we know, in the auricles. (See our remarks, in a future page, on Aneurism of the Heart.)

Inflammation of the substance of the heart always gives rise to dilatation of the cavity implicated, and this dilatation is proportional to the extent of the inflammation and to the number of its centres. When combined in an early stage with endocarditis it occasionally results in the formation of an *acute aneurism of the heart* (of which we shall subsequently speak), in consequence of a laceration of the tissue which has been loosened by the process of inflammation. Finally, as we have already remarked, centres of inflammation are not unfrequently the cause of spontaneous ruptures of the heart.

This affection commonly results in *induration and in suppuration*, although it much more frequently assumes the former than the latter mode of termination.

In the *former* we find, in place of the muscular substance, a white fibroid (cellulo-fibrous) tissue, either in the form of small stripes, or spread over a more extended surface, according to the size of the centres of inflammation and the mass of the inflammatory product; or we may observe, where the indurated product of inflammation is accumulated in larger quantities at definite points and forms a tissue of this nature, nodular, roundish or irregularly shaped, ramified tumors, having the toughness of callus, which protrude either externally, or internally into the cavity of the heart. This form of striped indurations is frequently found to be deposited in the same subject in great quantity on the most different strata of the muscular substance of the heart, especially where an accurate investigation shows us the residua of pre-existing endocarditis, combined with consecutive dilatations and hypertrophy. Professor Bochdalek has drawn attention to this fact and to the frequency of carditis, which has hitherto been overlooked and generally denied.

The more widely extended inflammations of the muscular substance of the heart exhibiting this termination are of especial importance. They affect either the inner layers of the walls of the heart, together with the trabeculæ and the base of the papillary muscles, including the endocardium; or the external layers, together with the pericardium; or, lastly, the wall of the heart throughout its whole thickness, including both the pericardium and the endocardium. Occasionally we find that contiguous portions of the innermost, the middle, and the external layers of the muscular substance of the heart, are in turn attacked. The muscular substance is here found to be replaced by a fibroid tissue, while the walls of the heart, the trabeculæ, and papillary muscles, appear to be converted into a white callous tissue;—a process in which the endocardium so far participates, that it not only enters to a corresponding extent into the same metamorphosis, and becomes identified with this tissue; but it even generally exhibits a gradually decreasing fibroid thickening beyond the limits of the metamorphosis in the muscular substance. We also observe at the pericardium exudations, which are either well defined, or spread over the whole heart, and have been converted into cellular or fibrous tissues; and these give rise to adhesions.

These generally-diffused metamorphoses, which affect the wall of the heart throughout its whole thickness, not only exert an influence, in a general sense, on the increase of the dilatation of the respective cavities of the heart by means of the inflammatory process, but also specially on the origin of defined saccular dilatations—*true chronic aneurism of the heart*—which we shall subsequently consider more at large.

The fibroid tissue in the wall of the heart, in the trabeculæ, and in the papillary muscles, becomes, not unfrequently, in the course of time, the seat of calcareous deposit, constituting what is termed *ossification of the walls of the heart*, which invariably depends on the pre-existing alterations of texture of the muscular substance of the heart, which we just described.

The termination of carditis in *Suppuration*, which is much less frequent, gives rise to *Abscess of the Heart.*

In accordance with what has been already stated, abscess of the heart is almost entirely confined to the wall of the left ventricle, where one or more accumulations of pus may be present.

They are generally of inconsiderable *size*, being about equal in circumference to a pea, a bean, or a hazel-nut. A more considerable size, if it does not consist in an extension of surface, is indeed incompatible with the continued existence of a recent abscess, since it would speedily be associated with a rupture of the walls.

These abscesses are usually of an irregular *form*, exhibiting various sinuosities, running in different directions.

The muscular substance of the heart immediately adjoining them, is in a condition of purulent infiltration and disintegration; at a somewhat greater distance, it is pale, permeated by a serous or sero-purulent exudation, soft, and admits of being easily torn; while still further from the abscess it is livid, and not unfrequently interspersed with varicose vessels; it is also relaxed. Occasionally, the contiguous muscular substance, in consequence of being infiltrated with a solidified fibrinous exudation, presents a lardaceous or lardaceo-callous appearance.

Under the last-named conditions, the abscess may be encysted, in which case it may exist for a longer period, while its contents may moreover become either in part absorbed, or in part condensed and cretified, and the abscess may in consequence be obliterated. Its usual termination, however, where paralysis of the heart does not supervene, will be *its opening* either internally or externally, and, in consequence, or independently of these causes, there will be complete *perforation* of the wall of the heart from laceration of the strata of the muscular substance, which are incapable of further resistance. It frequently happens in internal openings, that the endocardium not only suppurates, but is torn to an extent corresponding with the size of the abscess. Such an opening is followed by a discharge of pus into the cavity of the heart and its absorption into the blood; and very commonly, even before the symptoms of pyæmia have been fully developed, by a swelling of the muscular substance of the heart, owing to the penetration of blood into the cavity of the abscess, and by laceration of the remaining external layers of muscle, that is to say, by perforation.

Some very rare instances of superficial abscesses opening internally may be unattended by perforation, in which case the cavity of the abscess will constitute an acute form of aneurism of the heart, till the pyæmia induced by the discharge of pus into the cavity of the heart ultimately proves fatal. We are not acquainted with any well-attested case in which the discharge of pus has been restrained by the mass of the blood flowing into the opened cavity of the abscess, and by the deposition of fibrinous coagula, or where aneurism of the heart had, in this manner, become established for any length of time.

In the preceding remarks on endocarditis and carditis, and the sequelæ of these processes, we have frequently alluded to *Aneurism of the Heart*. The importance of this secondary heart-disease demands, however, that we should treat the subject specially; and we, therefore, purpose devoting the following section to its consideration.

Aneurism of the Heart, known also as partial (*Aneurisme du cœur faux*), or, according to Breschet, as *consecutive aneurism*, is a circumscribed dilatation of one of the cavities of the heart, depending specially on a diseased condition of the texture of the endocardium and of the muscular substance of the heart. We retain the designation of aneurism, with its inappropriate accompaniments of "*partial*," and "*false*," because the terms have been universally adopted, and because this condition exhibits in its pathological relations a certain resemblance to that which we designate *Aneurism of the Arteries*. We would, however, at once definitively explain, that we do not consider that there exists any close affinity between these two conditions. In fact, according to our views, this resemblance depends mainly on the circumstance that both conditions are based on an alteration of texture; we will, however, leave it to our readers to compare the two, and to analyze for themselves the special similarities and differences they may be found to present. We are utterly unable to concur in Thurnam's views on aneurism of the heart; nor can we adopt, as the sequel will show, the classification by which he divides aneurism of the heart into numerous species, corresponding to the different forms of aneurism of the arteries.

At the present day we are acquainted with only two essentially differing species of aneurism of the heart, one of which represents an *acute*, and the other a *chronic form;* the former corresponding generally to *false* and the latter to *true aneurism* of the arteries. We are led, from the numerous observations we have ourselves made, either wholly to discard all other forms, or at any rate to regard those as doubtful which are based on the unsatisfactory researches of other inquirers.

1. One, and certainly a rarer form of aneurism of the heart, is a proximate result of a recent inflammatory process of the endocardium, and probably, also, in great measure of the contiguous muscular substance of the heart, and depends on a laceration of the diseased tissue, which is itself the immediate consequence of its inflammatory relaxation. The blood rushes violently through the rent, which is either limited to the endocardium, or involves with it a portion of the adjacent layers of the muscular substance, and thus disturbs the still uninjured muscular tissue of the heart to various depths. A cavity is thus formed, whose walls consist of the upheaved, lacerated muscular substance, and which is surrounded at its mouth by a torn and fringed margin of endocardium. The blood poured into this cavity deposits its fibrin in the form of soft coagula, infiltrating the lacerated muscular substance, and occurring on the fringed membranous margin in the different forms of vegetations observed in the valves. This aneurism is developed in an *acute manner*, as may be seen from what has been already stated, and is accompanied by the appearances of recent endocarditis. We have never seen a case in which the walls of an aneurism of this nature had become consolidated into a fibroid, callous tissue; for, in all the cases we have examined, the aneurismal formation was only of recent date, having existed only for a very inconsiderable period after the endocarditis, during the continuance of which it had originated. None of the cases in which an aneurism with solid, callous walls, existed for any length of time after the endocarditis, afford the slightest evidence that it had originated in this acute manner from laceration. The investigations of foreign observers have so far influenced pathologists, that they have begun their inquiries regarding aneurism of the heart with callous walls with the preconceived opinion that a *læsio continui* occurs in the endocardium, as in the so-called *mixed aneurism* (*A. spurium* of Scarpa) of the arteries; and the difficulties attending the investigation of this form of aneurism of the heart, have greatly contributed to the maintenance of this error, notwithstanding the numerous proofs we have advanced to the contrary.

2. The second form of aneurism of the heart is either the remote consequence of the combined inflammation of the endocardium and of a somewhat thick layer of the muscular substance, or more frequently of inflammation of the wall of the heart throughout the whole of its thickness, accompanied with endocarditis and pericarditis. The inflammation of the muscular substance, by its tendency to induration, promotes the develop-

ment of a white fibroid tissue, which occupies the place of the muscular fibre in the trabeculæ, as well as in the actual muscular substance of the heart, and coalesces, as it were, on its inner surface, with the endocardium, which is thickened into a similar tissue, and towards the exterior, with cellular or fibroid formations,—the products of endocarditis and pericarditis. This tissue, with its inherent tendency to shrivelling, is unable to resist the pressure and flow of the current of the blood, and by its yielding and expanding gives rise to circumscribed dilatation of the cavity of the heart. The limits of this dilatation generally correspond with those of the metamorphosis of the muscular substance, extending as far as the point where the muscular fibre has remained undestroyed throughout the whole, or a considerable depth, of the thickness of the wall of the heart. The course of the development of this aneurism of the heart is therefore *chronic*, when considered as a remote result of the above-named combined inflammations.

It follows from the above observations, that this form of aneurism of the heart is a *circumscribed dilatation* of one of the cavities, whose walls consist wholly or for the most part of a *fibroid* (tendinous, ligamentous, cellulo-fibrous, callous) *tissue.* This circumscribed dilatation exhibits a *shallow sinus* in the muscular wall of the heart, or an ordinary *roundish sac*, or even a mere *appendage* to the heart, which communicates with the cavity, by means of an opening, corresponding in size to the cavity itself, or in some cases by a narrow aperture, or even by a short canal. This appendage either rises above the cavity of the heart from a broad basis, or rests upon it by means of a neck-like constriction. The *size* of the aneurism varies from that of a pea, a bean, or a nut, to that of a hen's-egg, or of the fist, or may be even larger. The form and size of these structures no doubt mainly depend on the extent and depth to which the metamorphosis of the muscular substance affects the walls of the heart, on their duration and locality, on the patency of the openings of the cavity of the heart, and on the original degree of tenseness and capacity for resistance in the walls of the aneurism. It is probable that the size of the aneurism will be the greater, and that it will the more nearly approximate to the form of a true sac or appendage, in proportion to the extent and penetration of the inflammation of the muscular substance, to its duration, its exposure to the action of the blood flowing into the heart and entering its own cavity through the action of the still uninjured muscular substance, to the degree of contraction affecting the openings in the respective cavities of the heart, and to the yielding of the adventitious tissue constituting the walls of the aneurism. Instead of coalescing with the pericardium, as is usually the case, large aneurisms have occasionally been met with, adhering directly to the thoracic wall and the lung.

The walls of this form of aneurism of the heart consist, as has been already remarked, of a fibroid tissue, which having taken the place of the muscular substance, coalesces internally with the thickened endocardium. The walls of this form of aneurism never present the slightest trace of a separation of continuity, either in the endocardium alone, or simultaneously in it and in one of the contiguous layers of the muscular substance; for the thickened investment of the aneurism occupying the place of the endocardium always extends beyond the boundaries of the aneurism to the normal wall of the heart, where it is gradually lost in the normal endocardium. The assumption that there is a *læsio continui* in the endocardium may have originated in the circumstance that the endocardium of the cavity in question is frequently found to be hypertrophied, that is to say, that it presents several newly-deposited layers, the most recent of which extend to the limits of the aneurism, which is filled with fibrinous coagula. The occurrence of an excessive morbid formation of the endocardium on the lining membrane of the vessels, from the blood, as shown in diseases of the arteries, must necessarily, from its great extent and importance, lead to future investigations. This form of aneurism of the heart corresponds to *true aneurism* of the arteries.

The walls of these aneurisms vary in thickness, although they are always thinner than the neighboring uninjured wall of the heart. They probably become so attenuated, in proportion to the increasing size of the aneurism, as to appear as if they were merely formed by the contact of the endocardium or pericardium, or of a doubled endocardium, in consequence of the aneurism having been developed towards another cavity of the heart. Osseous concretions, especially in the form of laminæ, are frequently developed in the tissue constituting the walls of the sac, whence the aneurism acquires a partially osseous character. (See p. 151.)

The cavity in this species of aneurism is very frequently filled with tough stratified fibrinous coagula, as in aneurism of the arteries. This, however, is usually the case only in larger aneurisms, and in fact the mass of fibrinous layers will, in general, be proportional to the size of the aneurism and to the extent to which the muscular fibres are destroyed.

The inner surface of these aneurisms of the heart occasionally exhibit the ordinary villous, shaggy, and warty, or even the so-called globular vegetations.

It is, moreover, worthy of remark, that new layers of endocardium are frequently found to be deposited in great numbers, and to a considerable thickness, upon the inner surface of the aneurism. It is only on a close inspection that they can be detected lying upon the subjacent fibroid tissue. The atheromatous disintegration they occasionally exhibit, imparts a certain degree of importance to their presence, since this atheromatous process is, in some instances, the cause of the origin of aneurism of the heart.

These two species, comprising an acute and chronic form, both of which depend on inflammation, embrace the numerous observations we ourselves have made, and will very probably, on an unbiassed inquiry, be found to include all cases on record.

That inflammation is the original controlling process in this affection is proved not only by the history of inflammation of the muscular tissue generally, and by that of the muscular substance of the heart in particular, but receives additional confirmation from the concurrence of the aneurism with endocarditis and its products, both as to their position and situation, even beyond the limits of the aneurism, generally even as far as the valves,—from the almost universal and simultaneous occurrence of the products of pericarditis,—from the nearly exclusive occurrence of aneurisms in those portions of the heart, which are in like manner the exclusive seat of carditis and endocarditis, viz., the left ventricle, —and from the residua of the secondary metastatic processes in the capillary system, which are frequently of simultaneous date with the endocarditis.

We see no grounds for concurring in the opinion of many observers who regard the alteration of texture of the endocardium and the substance of the heart on which the second form of aneurism depends as a peculiar or unintelligible alteration.

The question here arises, whether an abscess of the heart (*cardite ulcérative* of Bouillaud,) after opening into one of the cavities, can give rise to the formation of an aneurism. We are of opinion that aneurism of the heart does undoubtedly supervene, but the question is whether such an aneurism is of a persistent character. We have ourselves observed no case corroborative of such a view, and we doubt whether the pyæmia induced by the opening of the abscess could lead to the consolidation of its walls: that is to say, could heal the abscess by converting it into an aneurism. (Compare pp. 151, 152, on abscess of the heart, and the observations referring to the first form of aneurism of the heart.)

Moreover, we cannot suppose the so-called *atheromatous process*—as it occurs in the arteries—to be the primary condition giving rise to aneurism of the heart. We have already remarked, under the head of hypertrophy of the endocardium, that the newly-deposited layers of endocardium upon the wall of the heart have never, in any case that we have observed, been the seat of any but the earliest stages of that metamorphosis which terminates in atheromatous disintegration, while it is only in some instances that it is observed in a more advanced form in the valves. It is, however, worthy of remark, that we have certainly seen this atheromatous disintegration affect some portions of the depositions lining the inner wall of the aneurism, although in a form which proved that it could not, as a primary disease, have given rise to the formation of the aneurism, but must apparently have been subsequently developed in the already existing aneurism.

Aneurism of the heart *occurs almost exclusively* in the left side, and is incomparably more frequent in the left ventricle. There is only one undoubted case on record of this form of aneurism of the left auricle,—namely, that of Chassaignac, to which, however, we must add a preparation in our pathological museum of an aneurism of the acute form in the auricular septum. Hence these formations chiefly occur in the arterial half of the heart, which is known to be the almost exclusive seat of endocarditis and carditis. Like the above-named processes, aneurism rarely affects the right side of the heart: and the few cases on record of aneurism of the right ventricle, and those described as situated in the right auricle, prove (like those affecting the left auricle) not to be, strictly speaking, true aneurisms of the heart. These cases usually consist in a general dilatation of the auricle whose walls have been transformed by inflammation into a fibroid, callous, and even ossifying tissue of considerable thickness, and which itself adheres to the pericardium, having its cavity more or less completely filled up with fibrinous coagula.

In the left ventricle the apex is the ordinary seat of aneurism, and where it also attains its greatest size. It is less frequently observed towards the base of the ventricle, and is of very rare occurrence at the septum, which, however, is commonly more or less implicated in those cases where the apex is the main seat of the disease.

It is only in rare instances that we meet with more than *one* aneurism, and where two or even three exist simultaneously, they are generally in close proximity to one another, and not unfrequently present the appearance of one single aneurism, which has been more or less perfectly separated into two cavities by the marginal elevation of its walls.

That portion of the heart which is affected by aneurism is found, in almost all cases, to be also the seat of an active dilatation, occasioned by the cardo-endocarditic process and its sequelæ, by the aneurism itself, and simultaneous valvular affections.

The spontaneous rupture of an aneurism of the heart may be mentioned as an extremely rare *termination* of the disease. It may open into the cavity of the pericardium, into the pleura, or into the arterial trunk of the opposite cavity of the heart. Such an opening may occur in a chronic form of the disease as the final result of the increasing attenuation of the walls of the aneurismal sac in consequence of its own enlargement, as in a case we have observed where an aneurism in the ventricular septum, and near the apex of the heart, opened into the cavity of the right ventricle (the *varicose aneurism* of Thurnam). Acute aneurism of the heart more frequently terminates in laceration, which, in most cases, is very probably induced by the same cause which gave rise to the aneurism itself, namely, inflammation and inflammatory loosening of the tissue, and suppuration in the muscular substance of the heart.—In the case of a boy aged nine years, who presented extensive dilatation and hypertrophy of the left ventricle, and thickening of the endocardium at the septum, we observed immediately below the aortic valves, at the uppermost part of the septum, a laceration about the size of a pea, which led to a sac as large as a nut in the auricular septum, that had been formed by the upheaval of the muscular substance, and after penetrating into the right auricle, opened into its posterior side through an aperture about as large as a hemp-seed.

Most of the cases of aneurism of the heart have been observed in persons of mature age and of more advanced life. When, however, we bear in mind that by far the greater number of cases on record belong to the chronic form of aneurism, we are led to conclude that the disease of the tissue, which is the precursor of the aneurism, must have originated many years before the fatal termination of the disease, and therefore in an earlier period of life, the more so from the circumstance that an appreciable number of cases occur before the age of thirty, while we have observed the acute form in early childhood.

c. Metastasis in the Muscular Substance of the Heart.—Metastatic, purulent, and ichorous abscesses in the muscular substance of the heart may be reckoned among the ordinary conditions giving rise to metastasis, more especially when occurring in consequence of pus or ichor being taken up into the mass of the blood. There are usually several of these abscesses present, and they may result in laceration or ulcerous perforation of the heart. Metastatic processes are always simultaneously present, to a considerable extent, in other organs.

d. Gangrene of the Heart.—There is nothing, *à priori*, at variance with the possibility of the occurrence of gangrene in the muscular substance of the heart. Ulcerations accompanied with malignant products are not of rare occurrence, but the correctness of the observations purporting to refer to gangrene of the heart, have nevertheless been called in question by several writers, and we must remark that no case of the kind has fallen under our notice.

e. Adventitious Products.—Although adventitious products, with the exception of the adventitious tissue developed from inflammation, are generally of rare occurrence in the heart, yet some forms are not unfrequently met with; as, for instance, the varieties of adiposity of the heart, which we now proceed to notice.

1. *Adiposity of the Heart.*[1]—The occurrence of fat in the heart presents various anomalies, and exhibits different degrees and forms. (Compare the observations made on fatty degeneration of the Muscles, in Vol. III. pp. 239–241.)

a. The first form consists in the accumulation of an unusual quantity of fat on the surface of the heart. Fat is generally first abnormally deposited in those parts which, in their normal state, are covered by a certain quantity of fat, even in general emaciation; as the base of the heart, the sulcus transversalis, around the point of origin of both the arterial trunks, the sulcus longitudinalis and the course of the coronary vessels, the margin and anterior surface of the right ventricle, and the apex of the heart. The right side of the heart is always covered with large quantities of fat whenever there is any considerable tendency to the production of this tissue. In some cases this formation of fat is so excessive as to enclose the whole heart in a thick irregularly lobed mass of adipose matter, giving it the appearance of being enlarged.

This accumulation of fat in the heart is usually associated with a similar accumulation in the pericardium, in the mediastina, and in the abdomen (that is to say, in the omentum and mesentery, and on the gall-bladder), with fatty liver, and with general corpulence. The muscular wall of the heart, in young men of great muscular strength, is found to be in a normal condition, but in persons of advanced age, and in females, in conformity with the general character of the muscular substance, it is in general, relatively thinner, more flaccid, discolored, and paler.

The latter condition, which constitutes the transition to a second form of adiposity, and in like manner varies in degree, represents—

b. Actual Fatty Degeneration of the Heart,—Fatty Metamorphosis of the Muscular Substance.—The fat surrounding the heart penetrates inwards, and by gradually insinuating itself between the muscular fibres, tends in this way to displace the muscular substance. The apex of the heart and the right ventricle are especially subject to this form of degeneration, which, according to Laennec's observations, originates at the first of these points. When the left ventricle is implicated, the disease is usually limited to the apex, from whence it advances towards the right ventricle. It is only in its more intense stages that it affects the main part of the left ventricle. The muscular substance at the apex of each side of the heart, and consequently in the right ventricle, is frequently observed to be reduced to a layer, which, from its extreme thinness, scarcely admits of being measured, and appears like a mere muscular investment covering the fat. In cases of intense degeneration, the muscular wall of the left ventricle has even been found only from 2–1½ lines in thickness. The muscular substance is flabby and much relaxed, of a faded color, capable of being easily torn, and infiltrated with free fat. This displacement and disappearance of the muscular fibres is similar to the alterations observed in the muscular coat of the intestine when the mesentery is intensely fatty, and in the corresponding coat of the gall-bladder. The valves of the heart are at the same time thin and transparent, while the papillary tendons are softened.

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[1] Oesterr. Med. Jahrbücher, B. xxiv., St. 1.

This fatty metamorphosis does not only occur in the form of the above-mentioned transition stage, and in consequence of the excessive production of fat, and simultaneously with other accumulations of fat, but likewise independently of any such connection, and accompanied with general emaciation, as the result of tuberculosis and tuberculous phthisis, and lastly under circumstances that have not yet been explained. It is of frequent occurrence in conjunction with fatty liver. It is rarely met with before the age of 30–35 years, and is incomparably more frequent afterwards; it is also much more common in women than in men.

Considerable interest attaches itself to a not unfrequent combination of these two forms of adiposity of the heart, in which there is atheromatous disintegration and ossification of the morbidly deposited layers of the inner coat of the arteries, and especially of the trunk of the aorta, associated with aneurismal formation in the trunk of the aorta. These fatty degenerations are, however, very frequently associated with ossification of the coronary arteries,—a circumstance which will be further considered when we treat of these fatty accumulations on the trunk and extremities, attended with atrophy of the muscular substance, and with ossification of the arteries, which remind us of other analogous combinations of fatty accumulation with formation of bone, as in lipoma, fatty cysts in the ovaries, accumulation of cholesterin in ossifying cysts, &c.

However, we might be disposed to imagine that fatty metamorphosis would frequently terminate in spontaneous laceration of the heart, such is very rarely the case, even where the fatty degeneration extends to the left ventricle, which, as the ordinary seat of spontaneous lacerations, would seem predisposed to this lesion.

c. There is a third and very important, although hitherto unnoticed form (see vol. iii. p. 242) of this disease peculiar to the muscular substance of the heart, and differing entirely from the two previous forms of adiposity.

This form occurs more especially in hypertrophied and dilated hearts, in combination with the residua of endocarditis and carditis, or independently of these. The extent, seat, and duration of the disease present numerous remarkable diversities. In some cases, we observe scattered and distinct centres of inconsiderable extent, where the muscular substance is pale, flaccid, of a dirty yellow color, and soft and friable, rather than admitting of being easily torn, as is usual in relaxation. In other cases these centres are very numerous, and are found scattered over the true substance of the heart, in tuberculæ, and in the papillary muscles. They are ill defined, their margins being indistinct or obliterated. The discoloration presents a striped appearance as it follows the course of separate muscular fibres, decreasing in intensity from the centre outwards, and being finally lost in the normal color of the substance of the heart. This anomalous condition frequently extends over the whole inner layer of the muscular substance, which, when seen through the endocardium, after it has become thin and even transparent, presents the discoloration to which we have already referred, showing on a closer inspection, that this change of tint depends on the presence of fine yellow granules or globules, which are deposited in great numbers, in close contact, as if strung together on strings, in and upon the muscular substance, and variously entwined among the muscular fibres. The trabeculæ and the papillary muscles are usually diseased throughout their whole extent, as is also the muscular wall of the heart through its entire thickness, although not uniformly in all parts. This granular formation on and between the muscular fibres marks the intensity of the disease, which further corresponds with the degree of discoloration and softening of the muscular substance. A microscopic examination shows an accumulation of black and dark-outlined globules, which prove to be fat, while the muscular fibres are found to have lost their striated appearance, and the fibrilli are soft, and readily break down into delicate molecules.

This form of adiposity most commonly occurs in the muscular substance of the left ventricle, and, in cases of hypertrophy, also in the right ventricle.

This affection is, according to our observations, the most frequent cause of the *spontaneous laceration* of the hypertrophied left ventricle.

It may, moreover, probably be regarded as a consecutive disease of hypertrophy of the heart, since it is developed in consequence of the state of paralysis or inertness of certain portions of the muscular substance, induced by the disproportion between the mass of the tissues and the powers of innervation. The conditions of this disease are therefore similar to those experienced by the voluntary muscles in fatty metamorphosis (the second form of adiposity). We have occasionally observed this form in hypertrophied muscular membranes, when the paralytic habitus is established, as, for instance, in the hypertrophied muscular coats of the intestine and the bladder.

We have, however, occasionally met with this form of disease in non-hypertrophied hearts in young persons. Dilatation had probably been induced here by the adiposity, and the muscular substance relaxed in consequence. It is highly worthy of notice, that the papillary muscles are sometimes especially, and very extensively, diseased, as they may give rise to endocarditic murmurs and insufficiency of the valves, in consequence of inefficient action and tension.

2. *Cysts.*—These formations are very uncommon in the muscular substance of the heart, especially if we refer to cysts containing entozoa. In treating of them, we will limit ourselves to acephalocysts, deferring all notice of the cysticercus till we consider entozoa.

A sac containing acephalocysts is very rarely met with in the muscular substance of the heart, there being only a few cases on record, to which, however, we must subjoin two derived from our own observation. The parent sac contains either one or many *acephalocysts*. In one of the cases, we observed only one acephalocyst, which almost entirely filled up the cavity of the parent sac; while in the other, the parent sac, as far as the injured condition of its contents enabled us to judge, was filled with many of these cysts. We are induced, from the rarity of their occurrence, to give a short report of these cases, the former of which was rendered peculiarly interesting from the circumstance, that the presence of these acephalocysts occasioned sudden death. A short notice of the second case, in one of the medical journals, is the only account that has yet been published of either of these two cases.[1]

1st CASE.—This case refers to a young woman, aged 23 years, whose sudden death led to a judicial inquiry. The heart was somewhat enlarged and hypertrophied. The uppermost part of the ventricular septum presented a fibro-serous cyst with delicate walls, and larger than a hen's egg, which protruded into both ventricles, but more especially into the right and the conus arteriosus, and had so thoroughly displaced the muscular substance, as to be almost exposed. It had burst over an extent of 1½″ towards the right ventricle. From this opening an acephalocyst, nearly equal in volume to the parent sac, had been thrown with the blood into the conus arteriosus and the pulmonary artery, where it was found tightly wedged, and so far within the trunk of the artery as nearly to reach to its left branch.

The liver in this case was very large, and the right lobe contained *one* acephalocyst of the size of a child's head, and *two* of about the size of a hen's egg.

2d CASE.—The heart of a soldier, aged 35 years, was examined; his sudden death being made the subject of judicial inquiry, as in the former case. The posterior and uppermost part of the ventricular septum, and the contiguous portion of the posterior wall of the left ventricle, were occupied by a round sac of the size of a duck's egg, having callous walls of a line in thickness, which projected into the cavities of the right ventricle and auricle. Towards the back of the sac the muscular substance of the heart had disappeared while the heart itself was attached at that point to the pericardium by a dense cellular tissue. On making a section through the wall of the sac, a rust-colored stripe was observed between an outer and inner layer of white fibroid tissue, this stripe being the remains of the peripheral coagulum of a hemorrhagic exudation. The sac contained a pulpy brown fluid intermixed with crumbling and shaggy fibrinous coagula and the soft gelatinous remains of acephalocysts.

3. *Fibroid Tissue.*—Fibroid tissue very frequently occurs in the form of a fibroid thickening of the endocardium on the inner surface of the heart, as a fibroid thickening of the valves and their tendons, in the heart's walls, and in the tissue of the papillary muscles and the trabeculæ, where it is accumulated in different quantities and forms. We have always found that this product was based on some inflammatory process —endocarditis, carditis, or their combination.

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[1] Oesterr. med. Jahrbücher. Jahrg., 1841. Juni.

It appears in the muscular substance of the heart, either in the form of white stripes, of diffused strata of various thickness, or finally of large, roundish nodules, or irregularly branching masses. This substance, moreover, constitutes the greatest portion or the whole of the walls of aneurism of the heart, when of a chronic form.

We have never observed fibroid tissue in the heart under the form of an independent fibrous tumor.

4. *Anomalous Osseous Substance.*—Osseous structures are frequently found within and upon the heart in the form of bony concretions. They invariably originate in the fibroid tissue which is produced, as we have already mentioned, by inflammation, and in the deposition of new layers of endocardium. In this manner bony concretions are occasionally developed in the fleshy wall of the heart, and in the tissue of the papillary muscles and of the trabeculæ, in the form of nodular uneven plates, of nodular bands, or of irregular ramifying osseous masses. In the valves where these formations are of frequent occurrence, they are often in the form of nodular, ramifying bands and rings of different thickness. The whole fibroid mass sometimes ossifies, and may then be seen lying free and uncovered in the cavity of the heart, both in the valves and on the wall of the heart, when the muscular fibres are completely destroyed. Bony concretions in the valves are not unfrequently connected with others in the tissue of the heart, and (as we sometimes remark in diseases of the pericardium) with osseous formations in pericarditic exudations of fibroid texture; or lastly they form a bony mass, branching out in various directions. Smaller cylindrically-shaped concretions are often met with in the thickened tendons of the papillary muscles. In ossification of the valves these cylinders are larger and more connected.

The valves of the left side of the heart, especially the aortic valves, also exhibit osseous formations, which are developed in the morbidly deposited hypertrophied endocardium. In the auriculo-ventricular valves they form plates of inconsiderable size, and in the aortic valves string-like or nodularly rounded concretions. They are distinguished from the bony formations produced from the fibroid inflammatory callus by their yellow color and their similarity to ossifications of the arteries.

We have never been able fully to satisfy ourselves in reference to this last-named osseous formation upon the endocardium of the wall of the heart.

Fibroid tissue and the bony concretions into which it is developed, when they are the remote consequence of inflammation of the endocardium or the tissue of the heart, are limited, like this process itself, almost exclusively to the left side of the heart. The few cases in which they are observed on the right side of the heart are probably those in which there is ossification of the valves. The occurrence of this osseous formation in the left side is moreover limited to the valves and the ventricle, and never extends, according to our observations, to the left auricle. When bony formations occur in an endocardium which presents a morbid deposit, they are almost exclusively limited to the left side of the heart.

5. *Tubercles.*—If we except those cases of tuberculosis which have originated in the neighboring tissues and have extended to the organic muscular coats of other structures, as the intestine, &c., tubercles occur

in the substance of the heart with the same rarity as in muscle generally. It is only in extreme degrees of tuberculosis that we have discovered one or more tuberculous masses in the muscular substance of the heart in addition to a tuberculous exudation on the pericardium. We must, however, here except those cases in which large tuberculous masses, exuded on the external investment of the heart, have gradually imbedded themselves in the outer layer of the muscular substance.

It is remarkable, considering the similarity of the process of exudation on the endocardium and on the inner coat of the vessels with that on serous membranes, that tuberculosis should not occur on the two first-named structures.

6. *Cancer.*—Cancer of the heart is an extremely rare disease, and its occurrence is, probably, invariably owing to a highly developed cancerous dyscrasia, or to the proximity of a cancerous formation, as for instance in the mediastinum. The form of cancer affecting the heart appears, as far as we know, to be limited to medullary cancer in its genuine type, or in the form of melanosis. It is developed under and in the external investment of the heart, in any portion of the fleshy walls, or immediately below the endocardium, protruding, according to its size, more or less extensively either inwards or outwards, or even in both directions, in the form of nodules and clumps.

We have observed a case of acute medullary cancer of the heart accompanied by very general acute cancerous formation, in the form of numerous, small, roundish nodules, seated in the innermost layers of the substance of the heart beneath the endocardium, and even upon it, somewhat in the manner of globular vegetations. The above general observations will show that this mode of formation of cancer must be regarded as a highly interesting form of disease of the fibrin.

Cruveilhier (Livr. 29) has described the case of a ragged cancerous tumor seated on the inner surface of the right auricle, and projecting into the cava descendens and the right ventricle. This cancer probably resembled primary cancer of the veins.

7. *Entozoa.*—In addition to acephalocysts, to which we have already referred, the *Cysticercus* is by no means of rare occurrence in the heart, being then also simultaneously present in some of the voluntary muscles. There are seldom more than one or a very few of these worms to be found together in the substance of the heart; and in these cases they are also commonly present in the brain.

The Trichina, unlike the cysticercus, does not occur in the heart, although it exists in the voluntary muscles.

In addition to the above-named secondary formations we will here notice certain morbid structures which appear in the cavities of the heart, either free or adhering. These are not products of the endocardium, but essentially fibrinous concretions from the blood, and differing, therefore, from the above secondary formations, both in this and other respects. They constitute a series of formations which we will consider under the following title:

8. *Coagula, Polypi, Vegetations in the Cavities of the Heart.*[1]—The above terms have, at different times, been applied to this class of con-

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[1] Oesterr. med. Jahrbücher, B. xxiv. St. 1.

cretions. We refer our readers to the general remarks on the diseases of the blood and its fibrin for all that relates to the pathology, nature, and metamorphosis of these structures. We purpose here treating specially of their form, and shall only touch upon their other relations as far as is necessary towards the right comprehension of a subject which has been much beset with errors in the present day.

The structures now under consideration occur in many forms. The question here arises as to these structures generally, and each form specially, whether they are produced after death or before it, and how long they had subsisted during life. It has long been customary to distinguish certain fibrinous coagula from others by the designation of *death-polypi.* The fact that the blood, in consequence of the arrest of the heart's action, coagulates more or less perfectly in the cavities, into a loose soft clot, or a more compact mass, from which the fibrin is more or less thoroughly separated into a concretion, which, in its turn, exhibits the most various degrees of consistence or plasticity, has long been regarded as entirely in unison with the phenomena observed in drawn blood that has been left to stand and cool. The symptoms presented in the course of disease, the peculiar character of the phenomena exhibited in the death-struggle, the form of certain fibrinous structures in the heart, their relation to its inner surface, and their adhesion to the endocardium, have long since been advanced in support of the view that there may exist, during life, an independent self-persistent polypus of the heart. This view has continued, to our own day, to be so entirely misunderstood and misapplied that even ordinary death-polypi have very commonly been mistaken for true polypi of the heart.

No doubt can be entertained in the present day that fibrinous concretions are formed in the heart from the blood during life. It would appear certain that they form an organic (textural) connection with the inner wall of the heart; and further, that they experience various metamorphoses in their elementary composition. We purpose considering these in the sequel; but we would, in the first place, make a few general remarks on the conditions under which fibrin is separated during life from the blood, and coagulates into different forms of concretion. We will also consider, under the head of these respective forms, all that relates to their formation after death or during life.

These conditions exist partly in the heart and partly in the blood, and both are not unfrequently coexistent: the latter, however, are the more important, while the former are to be regarded as merely affording favorable momenta.

1. The first condition involves an abnormally prolonged continuance of the blood in the cavities of the heart in consequence of a decrease in the activity of the heart's action, as in hypertrophies of considerable intensity, passive dilatations, aneurisms of the heart, and in every death-struggle depending on general paralysis, or in consequence of pre-existing contractions (stenoses) of the ostia; or, lastly, there may exist various mechanical conditions in the form of inequalities and roughness on the inner surface of the wall of the heart and on the valves, in its passage over which the blood deposits its fibrin in a corresponding form.

2. The other and most essential condition consists in the tendency of the

blood to coagulate or to part with its fibrin in various forms of coagulation, either in consequence of spontaneous disease, or of the absorption in various ways, of some heterogeneous matter. Under this head we must especially class the so-called inflammatory (croupous) crasis, as it occurs in a primary or secondary form, associated with inflammations, pneumonia, rheumatism, &c.; the poisoning of the blood by the absorption of the multifarious products of the inflammation of normal or abnormal tissue, which have been produced within the vascular system on the endocardium, on the lining coat of the vessels, or externally to the vascular system, having in the latter case reached the blood by the most various channels.

The coagula in the heart may be classed in the following order in reference to their form. Many have only recently been recognized and duly characterized as fibrinous concretions, and these have received designations corresponding to their forms.

a. Clotty, roundish, membranous, ramifying coagula, when occurring in the cavities of the heart, commonly receive the designation of *polypi* or *polypous coagula*. These are variously sized clots, presenting differences in the number and length of the ramifying appendices by which their rounded forms are modified. They consist of a dark or blackish-red clot, from which fibrin is separated at some portions of the periphery, but seldom from the interior; or they consist, for the most part, of fibrin which has absorbed a certain quantity of cruor and serum, and appears colored with various tinges of red, or, when free from these, it exhibits a pale and somewhat dense coagulation. These concretions are especially common in the right side of the heart, where they are found in large quantities, generally associated with a loose coagulum and fluid blood, and exhibiting coagula which have been formed during the last moments of life and after death. This form of coagulum does, however, undoubtedly occur at various periods before death. Without entering into a description of these coagula, the limits of which it is difficult to separate, from those of the first-named variety, we would merely remark that the following conditions favor their development during life:

1. When they are situated in the left half of the heart, especially when extending into the aorta and its branches.
2. When their ramifications extend into the ventricles, and their branches are entwined among the trabeculæ and the tendons of the papillary muscles.
3. When they exhibit the impression of the contiguous surface of the heart, as is especially manifested in the auricular appendage (the auricle proper).
4. When they adhere or coalesce with the inner surface of the heart —the endocardium.
5. When they consist of pure fibrin, and are at the same time tough and tenacious.
6. When they exhibit a dirty yellowish and greenish color, and are, moreover, opaque.
7. When they present small purulent foci or tuberculous concretions.
8. When any one of the associated diseases of the blood is developed.

These coagula cannot in themselves be regarded as symptoms of endocarditis where other essential evidences of the presence of this disease are wanting; and even where the latter are present, they cannot be considered as affording any direct proof of the existence of the endocarditic process, but simply of a pre-existing and spontaneous disease of the blood, depending, probably, on the absorption of endocarditic products.

It is incontestible, that these coagula not only adhere to the endocardium, as has been observed, but that they are also capable of entering into an organic or textural connection with the lining membrane of the heart, and thus vegetate independently and without the aid of a vascular system, as they are directly surrounded by the liquor sanguinis. On submitting the observations hitherto made on this subject to the severest criticism, we meet, amid a mass of erroneous and hasty conclusions, with some few cases which unquestionably belong to this class of coagula. These tumors are of various size; of a roundish, oval, cylindrical form, which appear attached to a broad or narrow base, as by a pedicle; they are of a sponge-like, or elastic and tough, consistence; they generally consist of a fibroid structure, but in some cases exhibit a soft texture, composed of membranes, covered with elementary granules and cellular nuclei, of delicate fibrilli, and of thick and even tubular fibres and amorphous coagula. Their color is red, yellowish-red, or white. When they are gradually receiving one or more coverings of newly-formed endocardium, which extends from them to the inner surface of the heart, they present the appearance of having been developed below the original endocardium, or, at all events, in the innermost layers of the muscular substance of the heart. Osseous and cretaceous concretions may be successively developed in them; and the free stony concretions which former observers have recorded as occurring in the cavities of the heart, were, doubtless, nothing more than loosened, liberated, fibrinous coagula, which have become ossified or cretified. We have never hitherto been able to detect vessels in them. (See Faber, Thomson, Vernois.)

It is probable, that these coagula are somewhat diminished by a process of solution upon their surface, before they acquire any decided texture and are covered by layers of endocardium, and that they thus lose their original form, which is probably an irregular one, and become round. Such a supposition seems to derive support from analogy with the disappearance of vegetation on the valves of the heart, the gradual rounding of the globular vegetations, and the diminution and disappearance of the plug in an artery after the application of a ligature.

b. Globular vegetations (*végétations globuleuses* of Laennec) in the cavities of the heart constitute a second form. The formations distinguished by this designation are generally round concretions, varying from the size of a pin's head to that of a nut, attached by means of ramifying, cylindrical, or flat appendages or bands which entwine themselves among the trabeculæ of the heart; and are of a more or less uniformly dirty, grayish-red, or white color. They are hollow in the interior, but contain, within a wall of irregular thickness, a dirty grayish-red, or even chocolate colored thickish fluid, resembling cream or pus, and which is occasionally of a dirty whitish or yellow color. One or more of these concretions very frequently burst, when the fluid may be seen effused into the cavity of the heart, and distributed over the recent coagula which have been formed either in the death-struggle or shortly after death; or it is found mixed with the fluid blood contained in the cavity. The band-like appendages which they throw out, are either solid, or softened and liquefied in their interior.

Besides the structures of this form, there are others belonging to the same class which exhibit different relations, being of an oval shape, somewhat like a wedge, and presenting a shaggy or villous appearance. They differ from those already named, by adhering directly and firmly to the endocardium.

Although we very commonly meet with these structures in the condition above described, this state is not the primary one in which they occur, but merely the result of a metamorphosis to which the fibrinous coagulum has been subjected, not only in its elementary character, but also in its external form. Cases may be occasionally met with in the course of a long-protracted series of observations, in which this metamorphosis may be followed through all its gradations.

The globular vegetation is originally a solid fibrinous coagulum of irregular form, which varies in color according to the number of blood-corpuscles it contains, from different shades of red to a reddish-white color. This coagulum gradually assumes a roundish form, probably in consequence of the outer portion being taken up in the blood in a finely comminuted state. The metamorphosis which it undergoes is very important, and begins as a softening disintegration or solution in the interior of the nucleus, from whence it extends towards the surface. This process is so far developed in the globular vegetations above described, that there only remains a peripheral layer, which encloses the dissolved part as in a capsule. The soft and diffluent mass consists, as has been already remarked, of a pulpy, cream-like fluid, very often resembling pus, and of a chocolate, or dirty brownish-red, reddish-gray, pale yellow, or whitish color. A similar metamorphosis affects the ramifying band-like coagula, proceeding from the vegetations when they become hollow. The same process is occasionally discernible in the central layers of those coagula of the first form which have arisen during life; we sometimes observe in these coagula a tendency to decomposition, both by their turbidity and opacity, their dirty yellow color, their extreme lacerability, and by the appearance of a turbid cream-like moisture when they are compressed and torn.

This metamorphosis of the fibrinous coagulum is, moreover, highly interesting, from the numerous and important analogies it presents. It is here undoubtedly dependent on disease of the fibrin, from which the coagulum itself is formed, as we have invariably observed in these globular vegetations only in cases in which the blood is in a state of dyscrasia, as in *croupous* processes, after typhus, in the pyæmia of phlebitis, in a similar condition of the blood in the course of tuberculous or cancerous disorganization, &c.

It is a remarkable circumstance that globular vegetations are almost always limited to the left ventricle, where they are attached in the manner already described to the apex and the contiguous parts. We have, however, observed a few exceptional cases in which globular vegetations were situated in the left auricle as well as in the right ventricle and auricle.

A proof of the part contributed to their formation and attachment by mechanical conditions is afforded by the fact of their being deposited in the apex of the left ventricle, and in the appendages of the auricles—in short in those parts of the cavities of the heart which are most favorable to stagnation of the blood. We have seen these formations in the cavity of an aneurism seated at the apex of the left ventricle, and also, together with the ordinary valvular vegetations, on the mitral and the aortic valves in endocarditis.

While, on the one hand, every fibrinous coagulum, when considered in reference to its most essential feature—its metamorphosis—may be transformed into a globular vegetation, and coagula of the first form may thus be converted into these globular structures, there can, on the other hand, scarcely be said to be any true limits between the globular vegetations and those on the valves of the heart. The latter not only very frequently assume the globular form, as will be seen from the following remarks, but valvular excrescences pass through the metamorphosis of globular vegetations without assuming this form.

c. The third form comprises all those coagula that have in recent times been distinguished under the collective designation of *vegetations of the valves of the heart.* These were *formerly* known as *sarcomatous, fungous, condylomatous excrescences of the valves of the heart,* and have derived especial importance in our own day in consequence of being commonly regarded as an infallible criterion of endocarditis. This form, which is more frequent than either of the others, also presents the greatest variety in reference to number, bulk, shape, mode of attachment, color, consistence, and internal composition.

The *form* of these vegetations is partly influenced by their mass or size.

Smaller vegetations occasionally exhibit a superficial *roughness,* only appreciable to the sight and touch on a close investigation, and which is produced by the presence of *fine granular or extremely delicate villous* structures on the endocardium of the valves.

When these structures are deposited upon one another in a finely granular form, they are more prominently visible on the surface of the valves.

They commonly present *a coarsely granular* or *villous* and finally a *shaggy* appearance, measure several lines in length, and are arranged either in rows of *rigid,* pointed, unyielding, *excrescences,* or soft, relaxed, and *pendent villi.*

They form *shaggy appendages, having a thick, club-like, free extremity;* or, when of a more considerable size, they form round, oval, or pyriform *pedicled excrescences.*

Lastly, when of considerable dimensions, they somewhat resemble condylomata, having a *cock's comb or mulberry-like appearance,* or they are irregularly *nodular,* and either broad or pedicled.

Partial reference has already been made to the *dimensions* of these vegetations, which vary from the size of a hemp-seed to that of a hazel-nut.

As we have already remarked, these structures may occur in very *small* or in very *large numbers.* In *the latter case* different forms and sizes are usually found associated together; at the same time, they are commonly spread over a considerable extent of surface.

Their *color*, consistence, and composition vary according to their age, and the quality of the fibrin from which they are formed. We shall, however, revert to this subject in the proper place.

Their principal *seat* is in the valvular apparatus; they attack the mitral as well as the aortic valves of the left side of the heart, and are generally remarkable for the number and size in which they are exhibited in all the different forms of this affection, to which we have already referred. They are, moreover, observed on the tendons of the papillary muscles—in any part of the inner surface of the heart (the endocardium of which is, in consequence, thickened and rendered opaque, while its surface presents an absence of smoothness),—in and upon the margins of any fissure of the endocardium or of the subjacent tissue—on the margin of a fissure in the valve—on the edge of acute aneurism of the heart—on the torn extremities of a papillary tendon—on the inner wall of chronic aneurism of the heart—and, lastly, even without the heart, on rough, ragged, and uneven spots on the inner surface of the arterial trunks.

They occur especially on the valves in small numbers, in the form of minute granular or villous depositions at the separate segments of the auriculo-ventricular valves, or on the nodules of the semilunar valves, and in their vicinity. They, moreover, in some cases form a granular, villous, or shaggy margin of varying breadth, near the free edge of the valve, which, inclining in a crescent-like form along the semilunar valves, follows the fibrous coat in the parenchyma of the valves. When occurring in great numbers, they occupy a considerable portion of the free margin of the valve, and, assuming every possible form, extend upwards over the whole valve to the endocardium of the auricle, and downwards to the tendons of the papillary muscles.

At other portions of the endocardium they commonly form granular or delicate villous deposits at the margin and in the vicinity of fissures, and most frequently near some exuberant quantity of large villous masses.

It is worthy of remark, that all these forms of vegetations follow the course of the blood-current in every direction. Where they exhibit a broader margin on the auriculo-ventricular valve, this margin forms a projecting angle, from whence it is rapidly deflected. When they form villous or larger masses, they incline at the auriculo-ventricular valve towards the ventricle, and at the semilunar valves towards the direction of the vessel. We must also observe, that they are always situated on the side of the valve which is turned towards the calibre of the implicated opening.

In reference to their *color*, they are, when newly formed, and at the commencement of their existence, usually of a pale blue or yellowish red color, less frequently dark red, and are either uniformly colored, or speckled and seamed. They gradually become pale, resembling faint yellow, faded, and thoroughly washed fibrin; frequently, however, they do not part with their hæmatin, which in its further metamorphoses gradually loses its color, assuming a brownish-red, rusty yeast-like tinge, by which the vegetation is permanently characterized. These structures usually exhibit the consistence of a fibrinous coagulum, varying in their degree of softness or hardness; thus they usually become harder in proportion to the increased paleness of their color, although in some rare cases they are soft, dissolving like the globular vegetations.

On lifting or tearing off the vegetation, there immediately appears, if it be recent, a loosened, excoriated, and rough portion of the endocardium, which, in structures of older formation, is also raised and swelled up. As they become older, they at the same time become more firmly attached to the endocardium.

The following facts may be noticed in reference to their metamorphoses subsequent to the process from which they originate:

a. Vegetations once formed, in most cases, remain stationary for a long time, or even through the whole period of life, more especially when they have acquired any considerable dimensions; but it is certainly undeniable that they may, in the course of time, shrink and diminish, and exhibit an increase of condensation and consistence (*excroissances cornées, cartilagineuses*, Bouillaud), as we see in other fibrinous coagula.

b. There is no doubt that they diminish in a different manner, and that at times their presence is scarcely perceptible, since they often degenerate into fine, whitish, brush-like fibrinous villi, and in some cases even wholly disappear, without leaving any trace of their existence. The latter is proved by the circumstance (see p. 144) that, while in obsolete cases of endocarditis, the valves exhibit very insignificant or even no traces of vegetations, notwithstanding that they bear the impression of former intense disease, recent endocarditis very commonly presents a large number of these structures, characterized, in many cases, by the size and quantity in which they occur. These vegetations present an analogy with other fibrinous coagula within the vascular system, by being worn out, as it were, superficially, that is to say, they are taken up into the blood in fine particles, and are thus gradually diminished. This remark especially refers to such vegetations or portions of them as are separated from the fibrin of the blood of the heart in the form of coagula, whilst those which have been deposited by exudation remain and shrivel up. (See our subsequent remarks on the origin and nature of these vegetations.)

c. These vegetations on the valves—in perfect analogy with other fibrinous coagula—undergo, although less directly, a bony and chalky metamorphosis, constituting a special form of valvular ossification, to which we will revert in the sequel.

d. These vegetations seldom, and indeed never, unless when of considerable dimensions, experience that metamorphosis of softening, by which a fibrinous coagulum is converted into a hollow globular vegetation. This metamorphosis, which occurs in the early stages of recent vegetation, is undoubtedly the result of extensive diseases of the fibrin.

In conformity with their elementary character they consist, according to their respective ages, of elementary granules, cell-nuclei, and cells—of a homogeneous base, intersected by nucleated fibres, in the manner of the longitudinal fibrous coat—of fibres and fibrillæ resembling cellular tissue, and of thick tubular fibres.

The corresponding opening is more or less closed, in proportion to their number and volume.

In all considerations that relate to the origin of these vegetations we ought, in the first place, to notice their relation to the endocarditic process.

In the greater number of cases these structures are accompanied with the phenomena of endocarditis—the alterations of texture to which it gives rise; their appearance so far coinciding with these phenomena, that recent vegetations are found simultaneously to occur with recent derangements of texture, and obsolete vegetations with inveterate disturbances of texture—the residua of endocarditis. The question here arises in relation to these cases, *are these vegetations endocarditic exudations? and if not, how can their origin depend upon the process of endocarditis?*

In some rare cases they are observed unaccompanied with any phenomena of endocarditis; and here it may be asked, *how is their origin to be explained, and on what does it mainly depend?*

The result yielded by very numerous and widely differing cases are as follows:

a. That these vegetations, when considered collectively, are in some cases, *direct products of inflammation*—that is to say, *exudations.*

b. That in the great majority of cases they are *only in part* to be regarded as inflammatory products, since it is only the lowest layer, directly adhering to the excoriated valve, that can be considered in the light of an exudation, whilst *the greater number have been produced in another indirect and secondary manner from the endocarditis.*

c. That they also occasionally occur *without the existence of endocarditis.*

In the two latter cases, the vegetations occur as fibrinous coagula deposited by the blood, and their formation is effected in the following indirect and secondary manner:

Endocarditis induces a diseased condition of the blood, in consequence of the latter taking up its exudations. This morbid state is manifested by the readiness with which its fibrin coagulates and separates. As such coagula occur in different parts of the capillary system (as secondary processes in the spleen and kidneys), so also is the fibrin separated from the blood in the heart with a readiness proportional to the vegetations produced by the endocarditic process in the form of exudations, or the number of loose, rough, felt-like excoriated spots on the endocardium, either of which may exert a mechanical action.

The number and dimensions of these secondary vegetations accord with the intensity of this disease of the blood, and more especially with its character; and we find that they occur in the most exuberant masses when there is intense endocarditis, manifested by simultaneous disturbances of texture, and still more so where the disease is characterized by suppuration. This correspondence is further manifested in a remarkable manner by the great number of secondary processes in the different parenchymatous structures to which we have referred. The mechanical influence is more developed in proportion to the greater intensity of the endocarditic process. The number of vegetations is, however, most remarkable on the margins of a fissure in the endocardium and in the subjacent tissues, occasioned by inflammatory loosening of the texture, or still more, perhaps, by suppuration. It is obvious that the number of these vegetations increases with the extension of the endocarditis, and of the space over which the mechanical influence has diffused itself.

The metamorphosis of all these vegetations generally, and of those of the second form especially, depends upon an internal cause (namely the blood). Where, as is usually the case, the product deposited is of a benignant nature, the fibrin constituting these vegetations experiences the above-named favorable modifications, that is to say, the vegetations become condensed, gradually diminish, and even wholly disappear, or cretify. This is in accordance with such terminations as shrivelling, obliteration, and atrophy, which usually characterize the secondary process accompanying such an endocarditis. In some less frequent cases the vegetations undergo a softening process, and become diffluent in their interior, yielding a variously colored purulent fluid. This is observed in intense endocarditis, and when it occurs with purulent exudation, and in this respect it also agrees with those secondary processes of a less benignant character which terminate in purulent fusion. These metamorphoses are more commonly manifested in vegetations consisting of a large club-like villi or roundish masses, which, as we have already observed, accompany intense endocarditis, characterized by purulent exudations. The vegetations that begin to dissolve at the centre approach more nearly to the character of the *globular* kind in proportion to the roundness of their form.

The size and the metamorphosis of the vegetations afford evidence of the intensity of the endocarditis, and more especially of the quality of its products, when, besides these, other essential phenomena of endocarditis are present, and when the diseased condition of the blood can alone be referred to endocarditis.

In some rare cases where these vegetations are unaccompanied by any other important phenomena of endocarditis, they are usually inconsiderable in number and dimensions; and the question might arise, whether they may not even here originate in some very slight degree of endocarditis, which might produce scarcely perceptible disturbances of texture, that had been masked and hidden by the presence of the vegetations. We must, however, bear in mind that the origin of these fibrinous coagula is, in general, mainly dependent on some peculiar character in the blood, that it admits not unfrequently of being referred to some process remote from the heart, and that in some cases even it may be said to be spontaneously developed; that in addition to benignant and inconsiderable vegetations, there are other extensively diffused secondary processes in the different parenchymatous structures, which have a wholly heterogeneous character, and terminate in purulent fusion; that there is no trace of endocarditis to be detected, or, at all events, no new endocarditis corresponding to the recent condition of the vegetations; that a mechanical influence is especially important in the deposition of these vegetations which are formed not only on every rough part of the endocardium and the valves, but even on the lining membrane of the vascular trunks; and lastly, that the normal valvular apparatus, by means of its tendons, affords a highly favoring requirement for the separation of the fibrin. The above observations leave no doubt that, like other fibrinous coagula, these vegetations may be formed and deposited independently of simultaneous endocarditis, and in consequence of some other disease of the blood, upon any favorable portion of the inner surface of the heart, as, for instance, the free margin of the valves, which has become suited to its reception by incidental roughness or inequality of surface.

These vegetations cannot, therefore, be regarded as constituting an absolute indication of endocarditis, whose existence requires to be confirmed by the presence of more essential disturbances of texture, but must be considered simply as evidences of a diseased condition of the blood. If, however, such disturbances are present, these vegetations enable us, in the manner already described, to form an opinion in reference to the intensity of the endocarditic process, and the nature of its products.

ABNORMAL CONDITIONS OF THE VALVES, AND ESPECIALLY OF THEIR OSTIA.

§ 1. *Deficient and excessive formation.*

We have already considered this subject at p. 117, where we treated of the most important anomalies.

§ 2. *Anomalies of size,—Hypertrophy and Atrophy of the Valves.*

Anomalies of size in the valves, that is to say, their *superficial enlargement* or *diminution*, usually correspond to an altered thickness of the valves, the former being commonly associated with attenuation, and the latter with thickening of the valves. Exceptions do, however, occasionally present themselves.

Hypertrophy of the valves is found to be almost constantly associated with dilatation of the ostia of the heart, and here we see a healing tendency in nature which endeavors to maintain the valves in a state of sufficiency. We observe this in the auriculo-ventricular, as well as the arterial valves, and more especially in those upon the left side of the heart, which, as is well known, is more frequently affected with dilatation of the cavities and ostia. The valves, as we have already remarked, are in these cases usually thin, delicate, and transparent, and so attenuated as occasionally to exhibit actual perforations (atrophy); in like manner the papillary tendons are found to be thinner and more slender in proportion to the extent of the dilatation, while there is a striking thinness and transparency of the whole of the inner lining of the heart.—Exceptions are, however, occasionally observed; the enlarged valve appearing tolerably thick in comparison with the degree of its hypertrophy, which shows that the fibrous tissue of which it is composed must have increased in bulk. This is especially shown in hypertrophied aortic valves by the corresponding enlargement in size and thickness of their nodules, and the fibres passing from them.

If we except the shrivelling of the valves induced by the inflammatory process and its products, *atrophy* of the valves is of rare occurrence, although it may, indeed, very frequently be overlooked. It occurs in diminution (concentric atrophy) of the heart, and is manifested in the form of a shrivelling of the valves, more especially at their free margin, whence the whole valve, including the margin, is found to be thicker and less transparent.—Kingston has observed a case of shortening of the auriculo-ventricular valves, with unaltered thickness, flexibility, and transparency, and with normal width of the ostium, and has described it as a form of *atrophy of the valves*. Shortening may affect one, or more, or all the apices of the valves, and its immediate consequence is insufficiency. It has hitherto only been observed in the auriculo-ventricular valves.

Hypertrophy of the valves affects either their *fibrous texture* or their *investment of endocardium*. We have already observed that *hypertrophy of the fibrous basis of the valves* is occasionally associated with their general hypertrophied condition. We, moreover, frequently notice in the auriculo-ventricular valves, and especially the mitral, both in individuals of advanced life and in young persons, a pale white, yellowish-white bulging, or thickening of the valve towards its free edge, or a series of bulgings at the insertions of the papillary tendons, which, however, do not interfere with the function of the valve. No osseous concretions are ever developed in this hypertrophied tissue of the valves. In young persons, we occasionally meet with a condition of this portion of the valvular structure, which very probably indicates incipient hypertrophy of the fibrous texture, the free edge appearing swollen, more especially at the insertions of the papillary tendons. This bulging is produced by a pale red, translucent, more or less gelatinous substance, effused into the texture of the valves, from which, as from a blastema, the fibrous tissue is developed. This substance is very commonly found to consist of a translucent, partly homogeneous, and partly indistinctly fibrous mass, in which are imbedded numerous cell-nuclei, and the so-called nucleated fibres. It may be observed in reference to the arterial valves, that hypertrophy of the aortic valves, more especially of their nodules, is not of very rare occurrence. This last-named condition is, however, less frequently observed.

Hypertrophy of the Endocardium is, on the other hand, both more frequent and more intense in the arterial valves, where it more especially affects the aortic valves, as might be expected, from the greater tendency of the left side of the heart and of the trunk of the aorta, to a similar condition of excess of growth in the endocardium and the lining arterial membrane. The valves become thicker in consequence of the deposition of new layers, and the aortic valves more especially at their nodules and free margin present an appearance of bulging; the protuberance being roundish or cylindrical in form, uneven and nodular, and having occasionally a somewhat prismatic or facetted character from the pressure which they mutually exert on one another. The valves thus coalesce with one another, and with the walls of the arteries, by means of prolonged depositions from their lateral insertions. This increase of bulk, which is intrinsically important, is rendered more so in consequence of its secondary effects. A shrivelling process, similar to that by which the arteries are analogously diseased, now affects the valves, which become thicker, full and rigid, and degenerate into a cylindrically formed swelling, and by this means on the one hand contract the ostium, and on the other become insufficient. A bony substance may also be developed in the deposited strata in the form of nodular, round, or band-like ossifications, equally important with those affecting the arteries; or, lastly, this deposit may exhibit (as when it affects the arteries) an atheromatous disintegration and loss of substance resembling an ulcerous process, which, in the same manner as the ossifications, may produce fibrous coagula in the form of granular, villous vegetations.

This form of hypertrophy of the valves and its so-called consecutive phenomena, occur only in their greatest intensity in the aortic valves, for the disease invariably exhibits an inferior degree of intensity when it affects the auriculo-ventricular valve on the left side of the heart. This disease is always associated with hypertrophy of the endocardium, and more especially with the deposition of new arterial membrane in the aorta. Although it is most common in advanced life, it does occasionally occur at the age of thirty, or even earlier, and gives rise to the insufficiency of the aortic valves, which is very often gradually and almost imperceptibly developed in persons of advanced life. It is not of endocarditic origin, although it is very often erroneously regarded as a consequence and residuum of endocarditis.[1]

Atrophy of the Valves.—This disease is manifested by attenuation, unusual delicacy and transparency of the valves, and in its more intense forms by the formation of apertures within them. We have already instanced a condition of attenuation of the valves, as the consequence of their hypertrophied state. We, moreover, observe attenuation of the auriculo-ventricular valves associated with excentric atrophies and adiposity of the heart. The more highly developed forms of atrophy, in which there are perforations in the valve, are only found in the arterial valves, and more especially in those of the aorta; we do not remember to have observed any case affecting the auriculo-ventricular valves, and we should indeed be disposed to regard this disease as exclusively belonging to the valves of the arteries, if Kingston had not seen a few (three) cases occurring in the former, two of which were in the tricuspid, and the other in the mitral valve. This perforated condition of the valves occurs almost invariably, associated with hypertrophy, in consequence of the dilatation of the corresponding ostium. These perforations are almost always situated near the free margin of the valves, and more especially near their insertion, where they originate, increasing in numbers as they spread towards the nodules of the valve. They are, at first, about the size of a scarcely appreciable pin-hole or of a poppy seed, but after gradually enlarging by the confluence of several into one, they finally attain the size of a grain of millet or a hemp-seed, or even of a pea. When several are present together, they impart a reticular broken appearance to the valve. The perforations are, moreover, surrounded by a smooth margin, and are never round, but oval, elliptical, or fissure-like, and their long axis is at right angles to the free margin of the valve. They are also generally bounded by the fibrous bundles of the valves, so that the atrophy, at least at first, attacks only the thinnest portions.

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[1] Dr. Löbl has been led, by clinical observations, to adopt the opinion, that a disease of the valves of the aorta, differing from endocarditis, does actually exist.

Besides considerable and appreciable attenuation of the valve, and in some cases even perforation, we occasionally find some portions, as, for instance, the free margin, the nodule, and the fibrous bundles passing from it, thickened or hypertrophied.

Perforation of the valves is not of importance unless the apertures are very large, or some among them are deep and seated in the middle of the valve, and cannot be closed by the approximation of the valves; very generally, too, the symptoms are influenced during life by the simultaneous occurrence of heart-disease, as, for instance, dilatation of the left ventricle, and occasionally by the insufficiency of the atrophied valves, that is to say, by their inconsiderable magnitude compared with the dilatation of the ostium. It is, moreover, very probable that attenuated valves, independently of all other conditions, give rise to a change in the sounds of the heart in consequence of diminished resistance, and modifications in the capacity for yielding sounds.

It is in the middle and advanced periods of life, more frequently than in any other, that we meet with atrophy of the valves, in the more intense form associated with perforation; there is thus, in this respect, a perfect harmony with the periods most prone to dilatation of the ostia, of which the aortic opening is the one more frequently diseased. These periods further correspond with the age at which we most frequently observe excessive depositions of new membrane in the trunk of the aorta, the so-called atheromatous process, and ossification with dilatation; and atrophy of the valves is not uncommonly associated with these diseased conditions of the aorta.

§ 3. *Anomalies of Form.*

Malformations of the valves are alike frequent and varied; but as they are not possessed of any intrinsic importance we do not deem it necessary to enter upon any classification of them. They will be found under their respective sections, and it will therefore suffice to observe, that the malformations affecting the valves in consequence of endocarditis and of hypertrophy of the endocardium, are the most frequent and the most important. The valves of the left side of the heart would appear, from the observations hitherto made, to be the more especial seat of these malformations, more particularly when affecting the endocardium.

§ 4. *Anomalies of Consistence.*

We need do no more than simply refer to the anomalous toughness and hardness of thickened or shrivelled valves, and to the decrease of consistence which accompanies inflammation of the tissue of the valves in the form of relaxation and lacerability, as this subject has already been treated of in a different form under the head of atrophy of the valves.

We would *here*, however, enter more fully into the consideration of a morbid condition of the valves hitherto but little observed, as we know no other place to which we could more appropriately refer the subject, when regarded in a scientific point of view. We allude to a diminution of consistence in the form of an abnormal softness and tendency to laceration of the valves,—an appearance of the greatest practical importance. This disease, when considered in a practical point of view, might be termed *a gelatinous condition of the valve.* The cases in which we have seen it have not been rare, but they were always limited to the valves of the left side of the heart. We find that the valve, either throughout its whole extent, or at individual portions is more yielding, softer, and more readily torn; the faint whitish color, and the gloss of the fibrous texture disappear, and are changed to a pale yellowish tinge, approaching here and there to a reddish hue, while the whole becomes translucent. The latter condition probably depends upon the gelatinous non-adhesive substance effused into the tissue of the valve; but yet it is difficult to comprehend how the other anomalies can be produced which we find in this condition of the valve. The tissue of the valve was always found to have disappeared wherever this gelatinous substance was present, and the valve itself, after the removal of this extraneous matter, was observed to be in a state of extreme attenuation or atrophy. The question here arises, is this gelatinous substance a new formation, —a blastema effused here in order to be metamorphosed into fibrous valvular tissue, and for the purpose of strengthening the atrophied valve, —or is it the softened, disintegrated fibrous tissue of the valve itself? The former view appears to us incomparably the more probable, and we are of opinion that this gelatinous substance is the same which, as we have already observed, presents itself in a more dense and tough state, and in the act of undergoing a metamorphosis into tissue, in hypertrophies of the valves. The valve which is rendered soft and lacerable by attenuation becomes still more so from the deposition of this gelatinous substance by which the remaining textural elements are forced asunder. The softness and tendency to laceration of the valve would thus appear to admit of explanation on a mechanical principle, and not on any actual softening process of the tissue.

This gelatinous condition occasionally produces lacerations, more especially of the valves of the aorta. These can be easily distinguished from the perforations already described as produced by atrophy, appearing either as true fringed rents passing lengthways through the valve from its free margin, as fissures in the middle of the valve, or as a laceration or detachment of the valve from its insertional margin.—The gelatinous condition of the valves must, therefore, be classed amongst the more important diseases affecting these structures. The diminished power of tension and resistance must necessarily occasion some modification of the heart's sounds.

This gelatinous condition of the valves undoubtedly admits of cure, since the gelatinous substance may be gradually converted into a fibrous tissue, and thus condensed, by which means the attenuated valve increases in bulk, and is enabled, if necessary, to enlarge and adapt itself to the size of the dilated ostium.

This condition occurs, at the same periods of life, and under the same circumstances, as atrophy of the valves, either with or without simultaneous hypertrophy.

§ 5. *Separations of Continuity.*

Separations of continuity occur under the forms of laceration of varying depth at any part of the valve, from the margin towards its insertion—as perforation of the valve at different parts more or less remote from the margin—and as a loosening of the valve at the margin of its insertion; and affect the auriculo-ventricular as well as the arterial valves. Commonly only one or other of these forms occurs; occasionally, however, several are present either in one or more of the valves. One very important form of *læsio continui*, which does not affect the valve throughout its whole thickness, but only one of the layers of endocardium and a certain portion of its fibrous tissue, is especially worthy of notice, since it constitutes the basis of *aneurism of the valves*, to which we shall refer more fully in the sequel.

These lacerations of the valves are occasioned by disease of the valvular tissues, arising chiefly from their gelatinous condition; next in frequency, by inflammation (endocarditis); and, lastly, by the loosening of the tissue which accompanies inflammation of the valves. Lacerations of the valves are not only highly important, from the circumstance that their existence presupposes a high degree of the diseases we have already named, but also from their giving rise to valvular insufficiency.

§ 6. *Diseases of Texture.*

To these belong:

a. Inflammation (*endocarditis*) *of the valves*, which is by far the most frequently observed. This disease is especially important from its results, that is to say, from the morbid changes of the valves to which it gives rise, and the various heart-diseases depending upon the latter alterations.

Endocarditis, as we have already observed, especially affects the valvular system, which in many cases is *alone* diseased, while in others, it participates in the endocarditis attacking other parts. The valves of the left side of the heart are especially subject to this disease, as we have already seen; and even where the valves on both sides are diseased, those on the right side are always affected in a very much less intense degree.

Inflammation of the valves, in very many cases, is limited to the free margin, whilst, in others, it extends from thence to a various extent towards the insertion of the valve, and not unfrequently attacks the insertion itself, extending to the endocardium of the cavities of the heart and to the tendons of the papillary muscles.

In addition to what has been stated in reference to endocarditis, the following short notice may suffice to explain the characteristics of this disease.

1. *Redness and Injection—Vascularity of the Fibrous Tissue of the Valves*—can only be observed in rare cases of recent endocarditis, for this condition has generally passed into exudation, and cannot be recognized in consequence of the products deposited in the tissue of the valve. Considerable difficulty, moreover, attends the discovery of vascularity, even in recent cases, since it is most frequently masked by the redness of the valves occasioned by imbibition.

2. *Opacity and Bulging of the Valve* are among the most prominent appearances, and depend upon the deposition of inflammatory products in the tissue of the valve. They attain considerable intensity, and are either limited to the free margin of the valve, or extend over a greater portion of it; in some cases the whole valve with its attached margin, or in others with the papillary tendon, is implicated. The endocardium of the valve at several spots loses its usual smoothness and lustre, and the whole has a rough pilous appearance.

3. There may be *Loosening of the Tissue of the Valve*, which, in intense inflammation, predisposes to laceration.

4. *An Inflammatory Product*, which, in addition to the exudation infiltrated into the tissue of the valve and effused and solidified upon its free surface, appears in recent cases as a pilous and granular coagulum in the form of vegetations, or as a membranous exudation having a free finely villous surface, beneath which the valve appears rough, felt-like, and excoriated. In cases of long standing, these products may often be more readily recognized in the form of a more or less stratified pseudomembrane, on which depend the thickening and the various forms of adhesions and coalescence of the valves.

5. *Vegetations*, as we have already seen, are deserving of attention, although they cannot be regarded as absolute characteristics of endocarditis.

In the course and as consequences of inflammation of the valves, we observe:

a. Occasional Laceration of the Valves in one or other of the abovenamed forms, or laceration of one or more of the papillary tendons; the margins of the rent here generally exhibit an exuberant quantity of vegetations. Laceration is an invariable evidence of the existence of a high degree of the inflammatory process.

b. Inflammation, giving rise to *a purulent product* and to *purulent fusion* (*suppuration*) *of the tissue* of the valve, is also not very rare. It may under certain conditions give rise to aneurism of the valves; and is distinguished by an exuberant production of vegetations, which may be considerably diffused, and very frequently undergo purulent disintegration.

c. The most common termination of inflammation of the valves is:

1. *Permanent thickening* of the valve, arising from the product which is deposited in the tissue and on the free surface, and becomes converted into fibroid tissue. The degree of rigidity attained either by the valve and the papillary tendons, or by the former alone, depends upon the extent of the inflammatory process. Contraction of the ostium, and insufficiency of the valve, are frequent results of this condition.

2. This anomaly is rendered more striking when the thickened valve is finally *shrivelled*. This *shrivelling* may occur either in the direction of the perpendicular diameter of the valve, or concentrically with the axis of the ostium. The former produces shortening of the valve, and at the same time insufficiency, and the latter contraction of the contiguous ostium. Both acquire importance in proportion to the extent to which the valve is inflamed, and the contraction is most considerable in the auriculo-ventricular valves, when the inflammation has extended to their margin of insertion.

Hence arise numerous *Malformations of the Valves and of the corresponding Ostia.* Thus the auriculo-ventricular valves, when their free margin and the papillary tendons have been thickened and shortened, present the appearance of a rigid funnel penetrating into the cavity of the ventricle, and exhibiting an elliptic fissure-like opening at the mitral valve and a triangular opening at the tricuspid valve. When the whole or the greater portion of the valve has been thickened and shrivelled, the auriculo-ventricular opening of the left side degenerates into a fissure or button-hole-like aperture surrounded by a rigid string-like ring, while that on the right side appears like a somewhat rounded triangular opening. The arterial valves degenerate into an annular protuberance around the ostium, being of regular height and thickness when the disease is of uniform extent, or irregular in consequence of inequalities in the subjacent surface. In some extreme cases they form a diaphragm inclining with the concavity of its sinus towards the heart and perforated in the centre by a small opening. These are often associated with—

3. *Adhesion, coalescence or fusion* of the different apices of the valve and of the tendons of the papillary muscles to a greater or less extent. These papillary tendons are often found to be fused together into one single or several thick, smoothly roundish rigid strings or bands; but it is only in rare cases that one or more of these apices adhere to the contiguous wall of the heart or of the vessel. It is obvious that such a condition must contribute to produce contraction of the valve on the one hand, and insufficiency of the ostium on the other.

The contractions of the ostia produced by these consecutive anomalies of the valves are, moreover, heightened by the vegetations which so commonly occur.

4. *Osseous concretions* are frequently and variously developed as a secondary disease in the newly-formed fibroid tissue of the valves. These are occasionally small, scattered, nodular and roundish, or larger nodular rough band-like formations, and at other times complete osseous rings surrounding the ostium. From these rings the formations diverge in various directions towards the inner part of the valve, passing outwards from the attached margin, where they come in contact with other concretions developed in a simultaneously occurring pericarditic fibroid exudation. They may also be occasionally connected with concretions in the contiguous wall of the heart, when, together with its endocardium, it had been the seat of inflammation.

These morbid metamorphoses of the valves are, as we have already observed, by far the most frequent originating causes of dilatations and hypertrophies of the heart. Inflammation of the valves and its results must not be confounded with hypertrophy of the valves,—with excessive endocarditic deposition on them and its metamorphoses.

By way of supplement to this subject we will now treat of the so-called *Aneurism of the Valves.*

Aneurism of the Valves.—Some writers (Thurnam) have applied this designation to a morbid condition of the valves, which has indeed some affinity with aneurism, more especially if we adopt Scarpa's theory of *spurious Aneurism* (the *mixed Aneurism* of others).

From our own observations, we should be led to divide aneurism of the valves into two forms, and to compare them with the two forms of aneurism of the heart we have already described, although they do not indeed strictly correspond to one another, since only *one* (namely the *acute*) form of the disease in the valves corresponds with acute aneurism of the wall of the heart, while we have observed *no form of aneurism of the valves* corresponding to *chronic* aneurism of the heart. We would, however, include under this head consolidated (cured) aneurism of the acute form, affecting a valve, whose continuity is still undestroyed, although it may present unimportant sinuosities, such as are sometimes observed at the mitral valve near the free edge. We have found these structures in only one case, together with the residua of endocarditis, at the aortic valves, and, if they were not consolidated aneurisms of the acute form, they must have been produced by a hernia of one of the lamellæ of endocardium, through the fibrous layer of the valve.—This class would necessarily include the three cases described by Thurnam (one of which affected the mitral, one the tricuspid, and the third one of the aortic valves), since he regards them as dependent on gradual extension of the valves.

The following is *the mode of origin of acute aneurism of the valves*, according to the observations made by ourselves and others. In the course of intense inflammation of the valves, a *læsio continui* is produced in the valve, affecting only one of the laminæ of endocardium, and a layer of the contiguous fibrous tissue.

1. This *læsio continui* appears either as a separation or fissure of the structure, and may occur,

a. As the consequence of the condition of loosening and lacerability induced by inflammation,

b. Or it may arise from the loosened condition of the tissue, observed in the neighborhood of an abscess, in the parenchyma of the valve.

2. A *læsio continui* may also be the result of an abscess proceeding from the lowest part of the valve, and penetrating towards or even through its endocardium, or in other words it may be owing to a final suppuration of the endocardium. (In one specimen in our collection, a sinus even passes from an abscess in the substance of the heart towards the aortic portion of the mitral valve; above this the valve is torn from the ventricle, and the whole sinus thus converted into an aneurismal sac.) It is however very questionable whether the endocardium is actually in a state of suppuration in such cases, or whether it may not rather be lacerated above the adjoining abscess, in which case the whole process would essentially belong to that which has been considered under *b.*

When there is a tendency to laceration of the valve, this will occur with a frequency proportional to the extension of the aneurism in the direction towards which the blood flows to the valve,—the auriculo-ventricular valves being lacerated in the direction of the auricle and the arterial valves in that of the ventricle,—and especially when the *læsio continui* affects the surface of the valve against which the blood is propelled.

Thus where the valve has been perforated to a greater or less extent, the blood which impinges on it, penetrates into its parenchyma, and causes more or less extensive infiltration. By this means the yet uninjured portion of the valve assumes the appearance of a projecting tumor on the corresponding surface; and becoming, as it were, inflated, constitutes *valvular aneurism*, in the form in which we have observed it, and to which the following remarks apply.

This tumor is usually about the size of a pea or a bean, although, after continued attenuation of the layer of the valvular tissue of which it consists, it becomes as large as a hazel-nut, or even a pigeon's egg. The tumor is especially capable of such an enlargement at the auriculo-ventricular valves, in consequence of the more abundant mass of the fibrous tissue occurring in them.

Its *form* is round and hemispherical, or frequently so far irregular that it presents various sinuosities in the circumference of its base, as well as in its arched portion. It generally extends over a considerable space, in consequence of the widely diffused infiltration of the blood into the parenchyma of the valve.

Its *aperture* although originally a fissure-like rent, is generally round, and has fringed margins, which, together with the circumference of the valve, are covered with luxuriant vegetations.

Its *cavity* is filled with a variously discolored bluish-red, reddish-gray, yellow-reddish, dirty white, solid, or more frequently loose, soft coagulum, which very often becomes disintegrated like the globular vegetations.

Aneurism of the valves is, therefore, as may be seen from what has been already stated, an *acute formation occasioned by a considerable degree of inflammation of the valves.*

These tumors in general *terminate* somewhat speedily in lacerations. This usually occurs in the more intense aneurisms of the auriculo-ventricular valve at the highest point of the aneurism, or at the summit of one of its various pouches, in the form of a small fissure-like rent, inclining from its circumference towards the opening, and having its margins speedily covered with vegetations.

Death does not follow from this mode of termination of the disease, but results from the endocarditic process and the corresponding disease, to which the latter gives rise in the blood.—In the rare cases in which this aneurism is consolidated (that is to say, where it has its opening and the walls of the cavity covered with membrane), it constitutes a chronic aneurism, and, like simple or hernial sinuosities, continues longer (see the cases recorded by Thurnam and others), and may become fatal through consecutive diseases in connection with other coexisting heart-affections. It may be observed, in reference to the size of such aneurismal pouches of the valves, that in one case seen by Thurnam the tumor had attained the unusual dimensions of a large walnut.

Our own experience coincides with that of most foreign observers, in having discovered this aneurism on the valves of the left side only,—a circumstance that corresponds with the relation of endocarditis to the same side of the heart. It is probably always more extensive at the auriculo-ventricular valve, on account of the greater development of the parenchyma in that structure whilst it is lacerated in the arterial valves soon after its formation, and may thus terminate in a large fissure.—Thurnam, as has been already observed, found aneurism of the tricuspid valve in a heart in which there was a communication between both ventricles. In this case there were four aneurismal pouches on the valve.

From the above observations it will easily be seen, that although aneurism of the valves possesses a scientific interest, it is not of much practical importance when considered either on its own account or in reference to the intense disease from which it arises.

b. Adventitious Structures.—These are almost entirely limited to the occurrence of fibroid tissue and anomalous osseous substance (ossification), both of which are of very frequent occurrence.

1. The *fibroid tissue* presents various anomalies in reference to the elements of which it is composed. Thus, for instance, as will be seen under their respective heads:

a. It is found to be abnormally developed in hypertrophy of the valves.

b. It occurs in excess in those products (exudations) of inflammation of the valve which are developed in the tissue as well as on its surface.

c. The endocardium, deposited in excess on the valve, usually undergoes some metamorphosis of this nature.

d. A similar metamorphic process is observed in reference to the vegetations of the valves.

2. *Osseous formation* occurs in various essentially different forms, to which little attention has hitherto been paid:

a. The fibroid tissue produced by the process of inflammation, occurs in the above-mentioned form of protuberant, roundish, and band-like concretions. They are originally developed in the inner part of the thickened and shrivelled valve, from whence they increase in circumference, owing to the continued ossification of the fibroid tissue, and at length come to view uncovered in different portions of the cavity of the heart. They are closely analogous to the ossifications of fibroid exudations found in serous membranes.

b. The endocardium abnormally deposited upon the valve becomes ossified. These concretions are very frequent at the aortic valves, and of rarer occurrence at the mitral valve. In the former case, they frequently attain considerable bulk; but, in the latter, they are merely small plates. They correspond with the ossifications of the inner lining membrane which is deposited in excess in the arteries, and are originally developed, like these, in the lowest and earliest strata, being denuded and coming to view when all have been ossified. Such are the ossifications frequently observed in advanced life which have no connection with pre-existing endocarditis, however they may be associated with endocarditic products.

c. In addition to these concretions there is a third variety,[1] which is highly interesting from the many analogies with which it is associated. It presents itself most frequently (more especially at the aortic valves) as an osseous concretion in a stalactitic form, or as a rough granular agglomeration. These calcareous formations constitute a metamorphosis or conversion of the vegetations on the valve into bony and chalky matter.

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[1] Oesterr. Jahr. B. xxiv., St. 1.

As might be expected, and in accordance with experience, they are frequently found to be associated with one or more of the two above-named forms (more especially, however, with the ossifications considered under *a*), which are developed in the valve after it has been thickened by inflammation. These stalactitic osseous masses occasion and promote the continued formation of new vegetations, and are consequently very commonly surrounded by them.

Even the normal tissue of the valve becomes of a dirty yellow, faded color in advanced life, and exhibits a layer of fat and calcareous salts in a finely comminuted form.

Besides these secondary processes, we will consider:

3. The *atheromatous disintegration* of newly-deposited endocardium as it commonly occurs in a low degree on the valves.

4. Finally, in rare cases, where the necessary constitutional conditions are present, the vegetations on the valves of the heart exhibit a *carcinomatous* character, the cancer being usually of the *medullary* kind.

Review of the Anomalies of the Valves, and more especially those producing Contraction of the Ostia and Insufficiency.

We have endeavored, in the foregoing observations, to indicate those cases in which an anomaly of the valve produces contraction of the corresponding ostium, or the causes by which the valve itself becomes insufficient.

The causes on which *contraction* of an ostium depends, the mode in which it is variously developed through thickening or rigidity of the valvular apparatus, vegetations, &c., and the manner in which it may finally give rise to consecutive heart-diseases, in the form of hypertrophy and dilatation, are alike self-evident. This contraction is frequently so considerable, that the diameter of the auriculo-ventricular opening, more especially on the left side, scarcely equals that of the nail of the little finger, or even of a goose-quill, while the arterial opening would not admit of the passage of anything larger than a crow-quill.

The condition of the valves known as *insufficiency*, has only been adequately considered by modern observers. By the term *insufficiency*, we understand that condition of the valves in which they are unable to close the ostium, and thus allow the blood to return or regurgitate into a cavity of the heart which would be isolated if the ostium were completely closed. In this way, the insufficiency of the auriculo-ventricular valves allows a portion of the blood to return from the ventricles into the auricles during the systole of the former, while the insufficiency of the arterial valves allows the blood to return into the ventricle during its diastole.

As might be expected, we frequently find that one and the same anomaly of the valves produces contraction of the ostium and insufficiency. The latter is especially owing to the following anomalies.

1. *A relative diminution in the size of the Valves with dilatation of the Ostia*, the degree of the former depending on the intensity of the latter. As we have observed, the valves in these cases are commonly enlarged at the expense of their thickness and power of resistance, and they may continue to remain sufficient when the ostia are very considerably dilated.

2. *Perforation of the Valve*, in consequence of atrophy. It must be very well marked before it can give rise to any considerable degree of insufficiency.

3. *Laceration of the Valve*, under various forms, in consequence of the gelatinous condition of the valve, or perhaps, still more from its inflammatory state. The degree of insufficiency is increased in proportion to the extent of the laceration.

In like manner, *laceration* of one or more of the papillary tendons produces *insufficiency* of the valves.

4. *Shrivelling and Shortening of the Valve and its Tendons*:—the valve does not close the ostium, in consequence of its rigidity or its insufficient length. This insufficiency in the case of the mitral valve is in general owing to well-marked endocarditis; in the aortic valves it is often very slowly developed, and in advanced life, it is generally owing to an excessive deposit on the endocardium. This is the most frequent form of insufficiency, and the one which attains the most considerable degree of intensity; it is usually attended with contraction of the ostium, owing to the rigid, thickened, and shrivelled state of the valve. It will be seen from what we have already stated, at p. 173, that it is only in very rare cases that insufficiency ensues in consequence of shortening of the valve depending on atrophy.

5. *Fusion of the Valves with one another, or their Coalescence with the wall of the Heart or Vessel*, generally induces a high degree of insufficiency in combination with the above-named conditions.

It will be easily understood, that not only carditis and its metamorphoses, but also fatty degeneration of the muscular substance of the heart, especially when seated in the papillary muscles, may induce insufficiency of the valves.

Insufficiency of the valves gives rise to the same heart-diseases as contraction of the ostia; but, as has been already observed in p. 129, it has not been clearly demonstrated whether it specially induces dilatation, and on the other hand whether the stenosis specially gives rise to hypertrophy.

SUPPLEMENT.

Cyanosis has so long constituted a special subject of anatomical inquiry, that our work would be incomplete were we to omit stating our views in reference to this affection, and the relation it bears to heart-diseases. We must however observe, that our opinions are not derived from a careful consideration of all the known cases of cyanosis of the heart, but are, properly speaking, the mere expression of the views we have adopted from personal observation, and from the study of a limited number of the cases reported by others (Morgagni, Ferrus, Louis, &c.)

A distinction is commonly made between cyanosis, arising from organic *heart-disease*, acquired in advanced periods of life, or from *diseases of the lungs*, and cyanosis depending upon *congenital malformations of the heart*. The latter form is specially designated *cardiac cyanosis*; but we shall see in the sequel that both forms are identical in origin and character.

The cause of cyanosis, when depending upon original malformation of the heart, has usually been sought in the admixture of the venous with the arterial blood, either in the ventricles, the auricles, or the trunks of the vessels; and this admixture, together with the cyanosis, has been supposed to arise from a deficiency in the septa between the cavities of the heart.

According to our view, cyanosis does not arise from an admixture of the venous and arterial blood, which is in many cases very problematical, and not unfrequently altogether impossible, but depends rather upon *an obstruction in the passage of the venous blood into the heart, and upon an overcharging of the venous system, which is either transient or habitual, according to the circumstances of the case, and induces a corresponding repletion of the capillaries.* We moreover consider that all cyanoses generally admit of being classed under one head, however the causes from which they immediately arise may differ in depending on original and congenital, or acquired anomalies of the heart and lungs.

We are led to conjecture from our own experience, confirmed by the observations of others, that cyanosis never arises from malformations of the heart, consisting in deficiency of the septa, unless there exists at the same time some special anomaly of the arterial trunks, as narrowness or insufficiency of calibre, or contraction of the ostia of the heart. We will limit ourselves in the following notice to the most remarkable forms of this affection, and to cases which admit of being observed during a prolonged period after birth.

Patency of the Foramen ovale, although not uncommonly observed after death, is very generally not manifested by any symptoms during life, unless it occur in connection with some anomaly of the arterial trunks. This circumstance is the less remarkable when we remember that, under similar conditions, there may be an entire absence of the auricular septum, unaccompanied by the presence of cyanosis.

This patency cannot, in ordinary cases, be referred to any definite cause, and is, as far as we know, purely accidental; but in some comparatively rare instances, it certainly depends upon an anomaly of the arterial trunks, the patency of the ductus arteriosus, the presence of apertures in the ventricular septum, endocarditic metamorphosis of the valves, giving rise to contraction of the ostia in the fœtus, or upon pulmonary diseases, as catarrh, atelectasis, &c.

It must be observed, in reference to the question of a mixture of the venous and arterial blood, in patency of the foramen ovale, that in ordinary cases it is most probable that no such admixture actually occurs, inasmuch as the masses of the blood accumulated in the auricles equipoise one another, and the valve is pressed against the septum by the blood in the left auricle.

Symptoms of cyanosis do not occur even in cases of considerable deficiency of the valve of the Foramen ovale, without or even with the persistence of the fœtal condition of the Eustachian valve, which carries a portion of the blood of the Vena cava to the Foramen ovale, although in the latter case there is necessarily an admixture of venous and arterial blood.

In those cases, however, in which the patency exists conjointly with or is dependent upon the above-named anomalies, the symptoms of cyanosis are necessarily present, although this admixture of both kinds of blood is not invariably effected, as is commonly assumed, by the afflux of venous to arterial blood. The mode of admixture depends upon the nature of the accompanying anomaly in the vessels or heart. If for instance there is abnormal narrowness or obstruction of the pulmonary artery, the blood of the right auricle will be mixed with that of the left auricle in consequence of the obstruction to the escape of the blood from the right ventricle of the heart; but if, on the other hand, the aorta be the seat of the anomaly in question, the arterial will be carried to the venous blood. Either of these conditions will be induced in alterations of the ostia, occasioned by fœtal endocarditis, according as the right or the left side of the heart has been the seat of this process.

The patency of the Ductus arteriosus involves patency of the Foramen ovale from the right auricle, although not always in the manner usually assumed. It is supposed that the quantity of the blood in the left auricle diminishes with the width of the latter, as it flows into the aorta, and that a continued current of blood from the right auricle prevents the closure of the Foramen ovale. There are cases, however, in which the form of the open Ductus Botelli, and its two mouths, as, for instance, its expansion from the direction of the aorta, render it highly probable that the blood flows from the aorta towards the pulmonary artery, and, in such cases, the transmission of the blood of the right auricle, and the patency of the Foramen ovale, are the result of the excessive fulness of the former, arising from the passage of the aortic blood into the pulmonary artery, and the consequent obstruction to a free discharge of blood from the right side of the heart. In either case, whether the venous blood passes into the arterial, or the arterial blood into the venous, the presence of cyanosis will occasionally be manifested, in consequence of the inability of the blood in the Venæ cavæ to pass into the diseased heart when already in a state of dilatation.

Very considerable deficiency, or even the entire absence of the auricular septum, although necessarily accompanied with the admixture of the venous and arterial blood, does not give rise to cyanosis where the arterial trunks are normal. Numerous observations testify, however, that this deficiency very probably seldom exists unaccompanied by an anomaly of the vascular trunks, although its presence may frequently be overlooked. This consists in an obvious narrowness of the trunk of the aorta, which gives rise to a remarkable degree of cyanosis, although *the arterial blood obviously passes into the venous.* Narrowness of the trunk of the aorta, like contraction of the aortic opening, occasions active dilatation of the left ventricle, extending to the left auricle, and lastly, to the right side of the heart, through the capillary system of the lungs. The immediate consequence of the obstruction to the passage of the blood from the left ventricle and the auricle, occasioned by the narrowness of the trunk of the aorta, is undoubtedly to carry a portion of the arterial blood of the left towards the right auricle. A more remote consequence of the obstruction to the discharge of the blood from the left side of the heart, is to impede the passage of the blood of the Venæ cavæ into the right side of the heart, and we then have cyanosis as the result of the overloading of the capillaries from the Venæ cavæ.

It is evident that in these cases there will generally be a considerable degree of active dilatation of the right ventricle, especially of the Conus arteriosus and the trunk of the pulmonary artery. Bouillaud is unable to explain this circumstance, otherwise than by assuming that the right ventricle becomes arterialized from contact with the arterial blood, which enters it from the left auricle.

It would appear, from numerous observations, that *a deficiency of the ventricular septum*—as its perforation—and the communication consequently established between the two ventricles do not, in all probability, give rise to cyanosis, unless there exists a simultaneous anomaly of the arterial trunks. For, in the absence of this predisposing cause, and only under certain conditions, such as mental emotion, bodily exertion, or disease of the lungs, cyanotic symptoms are of rare occurrence and of a transient character. It must, however, be observed, that important anomalies of the vascular trunks are of such common occurrence with deficiency of the ventricular septum, that the latter condition is almost constantly associated with excessive cyanosis.

The anomalies of the vascular trunks most commonly associated with absence of the ventricular septum, are a more or less striking narrowness and obstruction, or even the complete closure of one or other of the arterial trunks, more especially the pulmonary artery, so that the aorta springing from both ventricles supplies the circulating system generally, and the lesser circulation especially, by means of anomalous pulmonary branches. The aorta here shows itself inadequate to the discharge of the blood from both ventricles, and the cyanosis must, therefore, undoubtedly arise from the obstruction opposed to the entrance of blood from the venous system, for we find in numerous cases of deficiency of the septum, where the vascular trunks are normal as well as where they are displaced, that cyanosis is either wholly absent, or that it occurs only on certain occasions, as, for instance, in pulmonary disease; that is to say, it arises in consequence of the retention of the blood in the venous system, by which the passage of the blood from the right side of the heart to the lungs is impeded; there can, however, be no doubt that an admixture of the venous and arterial blood is constantly taking place.

In like manner, where the entire ventricle is not properly developed, it and the vascular trunks to which it gives rise are rendered insufficient for the discharge of the mass of the blood.

The heart, in all these cases, exhibits dilatation and hypertrophy, which either affect both ventricles uniformly, or one more than the other, especially the right one, so that the heart retains its fœtal character, more especially in reference to the mutual relation of the ventricles.

Cyanosis is either continuous, although commonly remittent, or results from certain definite causes, among which we may reckon all those which influence the free passage of the blood through the lungs and heart,—as mental emotions, violent bodily exercise, &c. Pulmonary diseases may, perhaps, be regarded as the most powerful of any; and among these, the pulmonary catarrh which affects children and young persons is more especially influential in giving rise to symptoms of cyanosis; the more so, perhaps, because habitual bronchial catarrh is very commonly found to be associated with the above-named malformations, in consequence of the insufficient emptying of the pulmonary vessels into the heart. Cyanosis occasionally appears in childhood and puberty, when it is undoubtedly to be ascribed to a want of relation originating at this period of life, between one or other of the arterial trunks and the heart.

The appearances presented after death correspond with the character of the cyanosis, whether it be constant or transitory, and whether it have arisen from different known and obvious causes, or be owing to influences either unknown or unexplained; and we thus find that some persons suffering from cyanosis manifest retarded development, deficient nutrition and animal heat, and general debility, and die prematurely, while others exhibit merely a very slight depression of the functions of organic life. In some cases, in which the heart presented conditions admitting of the admixture of venous and arterial blood, all the functions were fully performed;—a circumstance that it has been attempted to explain by the assumption that no admixture of the two kinds of blood occurs, in consequence of the equal development of both sides of the heart.

A morbid form of growth frequently associated with cardiac cyanosis, is the drumstick-like or club-shaped form of the ends of the fingers, with a corresponding convexity of the nails. This phenomenon has not been explained, and if, as has been asserted by different observers, a similar malformation is acquired in pulmonary phthisis, it may serve, from its association with pulmonary cyanosis, to confirm our view of the mode of origin of cardiac cyanosis.

An important observation militating against the ordinary view of the mode of origin of cyanosis has been made by Breschet, who found, in one case, that the subclavian artery of the left side sprung from the pulmonary artery, while the extremities presented no anomalous color. We find, however, that there exists a species of local cyanosis in those cases in which the return of the venous blood has been obstructed by the afflux of arterial blood into a vein, as in varicose aneurism. Finally, in the fœtus there is no cyanotic color, although there is a constant admixture of the arterial and venous blood (Fouquier).

Capillary hæmorrhages of the most various organs constitute phenomena in every way important in cardiac cyanosis. They most commonly occur as bleedings from the lungs, and are undoubtedly occasioned by the rupture of the overcharged capillaries. They afford as strong a confirmation of our views as a case which fell under our notice of a cyanotic boy, aged 8 years, who died from laceration of the trunk of an insufficient aorta beyond its arch, and in whom there was an opening in the ventricular septum, closure of the pulmonary artery, and an origin of the aorta from both ventricles.

Cyanosis, or the abnormal formation of the heart on which it depends, may terminate in death, either suddenly and rapidly, or slowly, in the same manner as in acquired heart-diseases.

There is an anomalous form of cyanosis depending on *original narrowness of the arterial trunks, associated with a normal formation of the* *heart*, which constitutes a transition stage from the cyanosis, arising from malformations of the heart and of the arterial trunks to that form of cyanosis which is a symptom of acquired heart-disease. This anomalous narrowness, associated with a normal formation of the heart, extends in various degrees to the aortic trunk; and, like many other phenomena of cachexia, occurs most commonly during childhood and in puberty.

Finally, *cyanosis is a common symptom of many heart-diseases*, such as dilatations and hypertrophies of a higher degree, together with the anomalies of the valves, from which they originate. These diseases also commonly give rise to acquired anomalies of the vascular trunks, such as contraction and obliteration, acquired communications of the aorta with the pulmonary artery and with the Venæ cavæ, and the consequent entrance of arterial blood into the two last-named vessels. These forms of cyanosis very frequently do not appear decidedly until advanced periods of life, although the heart-disease may have been acquired in early childhood, if it be not even congenital. It is still problematical whether this form of cardiac cyanosis can be acquired by *the reopening of the closed Foramen ovale and by a morbid perforation of the septum, owing either to inflammation and suppuration, or to a fissure.* This view of the possibility of the reopening of the Foramen ovale originated at a period when too much importance was attached to its patency, and would appear to be especially designed to serve for the completion of the whole theory. The cases recorded of acquired morbid perforation of the septa are certainly not wholly improbable, but the previous history of these cases affords us no convincing grounds for the assumption of the process of inflammation and suppuration or the existence of a fissure. These cases are also incapable of solving the questions whether these processes may not date from the period of fœtal life, and whether, therefore, the morbid perforation may not be a congenital heart-disease; nor do they show whether the case may not be one in which the products and residua of inflammation belonged to a process subsequent to the perforation rather than to one by which this process was effected. This perforation deserves the more consideration, since traces of old or recent endocarditis have not unfrequently been found in hearts presenting such anomalies of formation.

Cyanosis may not only be derived from the heart (where it most commonly originates in the right side), but it may also depend on the most various congenital and acquired *diseases of the lungs*, which impede the circulation of the blood in the capillaries. The insufficiency of the pulmonary capillaries to carry the blood from the right side of the heart, causes an impediment in the discharge of the venous system into the right cavities of the heart, and thus gives rise to cyanosis; and it, moreover, as is clearly observed when of long continuance, induces active dilatation of the right side of the heart, the intensity of which corresponds to the degree of the impermeability of the pulmonary capillaries. We purpose in a future page to treat this subject more in detail.

Diseases of the left side of the heart, such as dilatation and hypertrophy of the ventricle, but more particularly contraction of the auriculo-ventricular opening, occasion the right side of the heart to be over-filled, in consequence of the obstruction opposed to the discharge of the blood from the capillaries of the lungs; and hence we have the phenomena of cyanosis, usually with extension of disease (dilatation and hypertrophy) to the right side of the heart.

Cyanotic phenomena of various degrees of intensity may also depend on conditions of excessive density, and on continued compression of the lung (as for instance from exudations), on atelectasis of the lung, on catarrh and bronchial dilatation, emphysema of the lung, extensive pneumonia and pneumonic induration, pulmonary tuberculosis, &c., in like manner as on narrowness and closing of the arterial trunk, and admit of an equally easy mode of explanation. These phenomena of cyanosis may, moreover, either when congenital or acquired soon after birth, obstruct or wholly prevent the involution (closure) of the fœtal passages.

All cyanoses, or rather all forms of disease of the heart, vessels, or lungs, inducing cyanosis of various kinds and degrees, are incompatible with tuberculosis, against which cyanosis offers a complete protection, and herein we find a key for the solution of the immunity against tuberculosis afforded by many conditions which at first sight appear to differ so widely from one another.

III.—ABNORMAL CONDITIONS OF THE ARTERIES.

§ 1. *Deficiency and Excess of Formation.*

We have already treated, in their connection with anomalies of the heart, of all anomalies or other defects of structure of the two arterial trunks, in so far as they present any true pathological interest. We would here only notice the following facts.

It is extremely doubtful whether there is a complete *absence* of a vascular system even in the most imperfect monsters, although extreme deficiency and a very imperfect indication of the two systems, is common in these cases. Where there is a deficiency of individual portions of the body there naturally exists some anomaly in the corresponding portions of the vascular system, which will present deficiencies corresponding in intensity to the degree of arrest of development.

Supernumerary parts present a corresponding *excess of formation* in the vessels, although it must be observed that as a multiplication of organs is not uncommonly merely apparent, the multiplication of the arteries and veins implicated in the anomaly is only apparent, indicating the mere cleavage or subdivision of a trunk. We have already spoken, and purpose treating more fully, of the excess of vessels supplying different new formations. To these *anomalies in the number* of the individual vessels belong:

§ 2. *Anomalies in the Origin, Course, &c., of the arteries.*

These, which are known as varieties, are very numerous, and are in part produced by anomalies of position and form in the corresponding organs. Several are highly important in reference to operative surgery; but as we are unable, from personal experience, to add any new facts in relation to this subject, we would refer our readers to the Monographs in which it has been treated, and to the ordinary Systems of Surgery.

§ 3. *Anomalies and Diseases of Texture.*

We commence with the consideration of these anomalies, because they are both intrinsically important, and constitute the foundation of the principal consecutive anomalies and diseases, and because a knowledge of them is absolutely necessary for the right comprehension of the great majority and the most important of the alterations affecting the calibre and thickness of the walls of the arteries, their separations of continuity, and the subsequent phenomena to which they give rise. We shall institute various comparisons with the corresponding textural diseases of the veins, and we would here specially refer, on all these points, to the remarks we shall have to make subsequently on the diseases of the veins.

a. Inflammation of the Arteries—Arteritis.—In the first place it will be necessary clearly to comprehend whether there actually exists a spontaneous *arteritis*, and whether that special form of *arteritis* ever occurs which is commonly supposed to be met with in our hospitals and dead-houses.

If under the term arteritis we understand an acute inflammation, in which the inflammatory products are deposited or exude, as in phlebitis (inflammation of the serous membranes, &c.), on the free surface of the lining membrane of the vessel—that form of inflammation which is supposed to give rise to those appearances of redness commonly observed after death in the inner coat of the arteries—it will be necessary to notice the following points in reference to this subject:

1. The absence of vessels in the (yellow) circular fibrous coat, and more especially in the inner coat of the vessels, forbids our assuming the possibility of inflammation in these layers. This is also fully confirmed by experience, and we find that the redness observed in these coats, more especially in the lining membrane of the vessels, is obviously owing to imbibition, which is developed after death, and possibly even during life, with a rapidity proportional to the state of decomposition of the blood. The coloration always proceeds from the inner surface of the vessel without a trace of vascularity, and penetrates to different depths in the yellow membrane; whilst there is no product of inflammation to be discovered either on the inner surface of the vessel or in the tissue of either of these arterial coats. The phenomena manifested during life by the supposed arteritis, are in such cases always dependent on primary or secondary disease of the blood.

2. The cellular sheath of the vessel is alone capable of inflammation, and we are here led to inquire, whether this inflammation can deposit its products on the inner surface of the artery, under what conditions this may be done, and, what experience teaches us in reference to this subject.

The circular fibrous coat in the larger arteries, as, for instance, in the trunk of the aorta, exhibits so great a thickness, together with such density of texture, that we are unable to comprehend how it can be permeated by an exudation, unless by the agency of an acute process. This is fully confirmed by experience; for, at all events, we have never detected any such process in the trunk of the aorta, or, in other words, never observed arteries in the sense in question; and we are, therefore, led to deny, or at all events to doubt the correctness of the observations recorded in reference to this subject. According to our view, the supposed pseudo-membranes on the inner surface of the aorta, and its redness, together with the fibrinous plug obstructing its canal, are separated and coagulated from the diseased blood.

We, moreover, regard it as very doubtful whether pus is ever produced in the inner coat of the arteries or between this and the middle one, nor do we attach any great weight to the observations of Andral, in which, as he asserts, he found some half dozen abscesses of the size of hazel-nuts under the inner coat of the aorta.

Experience alone is able satisfactorily to determine the limits at which the thickness and density of the texture of the circular fibrous coat cease to oppose an absolute obstacle to the imbibition and permeation of an exudation produced in the cellular sheath of the artery, or to its appearance on the inner surface of the vessel. We find, indeed, from observation, that such inflammation frequently exists in the *femoral arteries*, especially in women, and likewise in the *umbilical arteries of new-born infants;* and we are hence led to conclude, that it may occur in all those arteries generally which possess a like organization.

The *anatomical appearances of acute arteritis*, considered within the limits we have assigned to its occurrence, are:

1. *Injection, Redness of the cellular sheath of the vessel.*—This is commonly no longer distinctly apparent after the establishment of those products which we are about to name.

2. *Infiltration of the cellular sheath*, with a serous, sero-fibrinous, partially solidifying moisture, causing puffiness; in some few cases the tissue exhibits purulent exudations, which are either diffused, or limited to individual points, or grouped into circumscribed foci.

3. *Extreme lacerability—the facility with which the cellular sheath may be removed* from the circular fibrous coat.

Hitherto we have only enumerated the appearances attending inflammation of the cellular tissue. (See vol. iii. p. 18.)

4. The *circular fibrous coat* appears loosened and succulent; admits readily of being drawn into fibres and separated from the elastic coat; is commonly blanched, and sometimes colored in different shades of red from the inner surface of the vessel through imbibition, although without any apparent injection. *The lining membrane* of the vessel is loosened, and may easily be detached or torn; its free surface is dull, and occasionally exhibits a felt-like or wrinkled appearance; it is either pale or reddened through imbibition.

5. The vessel is frequently, although not always perceptibly *dilated*, which is owing to the paralysis of the elastic layer of the cellular sheath and the circular fibrous coat. A coagulum of blood acting as a plug is lodged in the canal of the vessel, whose bore is thus more or less completely filled and obstructed.

6. *The presence of a free product (exudation) covering the inner surface of the vessel in the form of a pseudo-membrane* is, in most cases, problematical. It occasionally occurs in the form of a soft, pale yellowish,

or yellow-reddish layer, differing from the inner lining membrane of the vessel, as well as from the peripheral stratum of the plug. In more frequent cases, that which may be regarded as a free product, is merely the outer layer of the plug in the act of being metamorphosed into a structure analogous to the inner coat of the vessel. In these cases, the exudation thrown out on the inner surface of the vessel is taken up into the blood before it has been consolidated and has thus given rise to the formation of an obstructing coagulum. In many cases a portion of the exudation is solidified under one of the strata forming the inner coat of the vessel—below the epithelium and the longitudinal fibrous coat—and these strata being thus loosened are thrown off. They form a covering to the plug which projects into the canal of the vessel.

These appearances constitute the so-called *adhesive arteritis.*

7. In rare cases arteritis gives rise to a partially or wholly *purulent exudation*, which may be recognized by the following appearances:

a. The inner surface of the vessel sometimes distinctly exhibits a thin layer of purulent exudation, which is partially attached to the plug.

b. This exudation, which is discolored and loosened, undergoes a process of softening, both in its interior and at different points of its circumference, and is reduced into a puriform semi-fluid mass, or into a fluid exhibiting a corresponding degree of decoloration.

c. The inner and the circular fibrous coats are swollen, unusually succulent, of a somewhat yellowish color, loosened and stratified, and are distinctly infiltrated and permeated with the purulent exudation.

d. The whole of the lining membrane of the umbilical arteries of new-born infants is frequently found to be detached from the yellow membrane, which again is separated from the elastic coat by means of the exudation which is produced from the cellular sheath, and is, for the most part, accumulated between these coats.

e. The cellular sheath exhibits the above-named signs of inflammation in a highly developed form, having purulent exudation diffused over the tissue or accumulated in circumscribed foci.

This form of arteritis deposits, therefore, as appears from the above observations, a product which is either capable of coagulation and solidification, and of being metamorphosed into tissue, or is of a purulent character. The first of these forms is commonly termed *adhesive*, on account of the obliteration of the vessel to which it very usually gives rise; it is of much greater frequency than the other form.

In reference to the modes of *termination* and the metamorphoses of the products of this form, we must notice the following particulars:

1. Adhesive arteritis occasionally passes into *resolution*, as does more frequently adhesive phlebitis; the products of inflammation in the tissue of the coats of the artery are resorbed, while the plug is gradually dissolved, and taken up into the blood in a finely comminuted condition.

2. *The ordinary termination is permanent and more or less complete obliteration, which, in its turn, gives rise to atrophy of the vessel.*—The plug in the vessel gradually shrivels, being decolorized and converted into a fibroid string. When the plug has entirely filled the vessel, and is attached to the inner wall by means of one of the above-named structures, the coats of the vessel will be found to adapt themselves to its

shrivelling, while the vessel closes around it into a solid cylinder. But where the plug has not completely filled the artery, and is only attached at some points to the wall of the vessel, or where the shrivelling and metamorphosis have been effected too rapidly to allow of the walls of the artery following the process uniformly, and the adhesions have therefore been drawn aside and partially loosened by the flow of blood, the obliteration will be incomplete. In these cases the artery is occupied by a fibroid cord or string, which closely adheres at certain points to the wall of the vessel, although free at all other parts, or is, at the same time, attached in different places by means of string-like structures or pseudo-membranous plates, so that the calibre is very much contracted, and the circulation, more especially in the smaller vessels, correspondingly impeded. In consequence of complete closure, the specific tissue of the artery, more especially the circular fibrous coat, very rapidly disappears, and the vessel becomes converted into a hard fibroid cord enveloped in cellular tissue.

Bony substance may be developed in this fibroid cord, which may be ossified over various extents of its surface.

3. Arteritis with purulent exudation leads, in some few cases, to complete or partial *suppuration—ulcerous destruction of the vessel.* This occasions hemorrhage, which, according to circumstances, is either external or directed into the tissue—a result which very rarely occurs in spontaneous arteritis, although frequent in the suppuration arising in an artery after it had been tied.

Arteritis is very rarely fatal through the fever by which it is accompanied, or the various inflammations to which it gives rise in important organs. Inflammation of the umbilical arteries, when it extends to the peritoneum, often proves fatal through peritonitis. Spontaneous gangrene in the form of mummification, commonly known as gangrena senilis, is often produced during arteritis or occurs as one of its sequelæ, when it attacks the trunk of a vessel belonging to a part of the body, which cannot be supplied by any collateral circulation. This result of arteritis, which is dependent on the closure and obliteration of the vessel, has been most frequently observed in the lower extremities in inflammation of the femoral arteries. It is a common cause of the fatal result of spontaneous arteritis.

General infection of the mass of the blood, as the consequence of the absorption of the products of arteritis into the blood, and of the secondary metastatic processes in the capillary system with which it is intimately connected, is, according to our experience, a *very unusual phenomenon*, and hence a very uncommon cause of the fatal termination of arteritis. Our own opinion is confirmed in reference to this point, by the concurrent testimony of other observers. (Hasse.)

We have already endeavored to explain this rarity of the secondary processes, as compared with their frequency in phlebitis. We will here briefly observe, that this rarity must be referred to the greater susceptibility of the arterial blood for taking up inflammatory products which speedily give rise to coagulation and to obturation of the vessel, and to the circumstance that their reaction in the arterial current, being exhausted towards the capillaries in ordinary cases, hinders the general infection of the blood beyond the limits of those vessels. The inflammation of the veins accompanying an inflamed artery, which we have had very frequent opportunities of observing, does not appear to us always to possess the character of secondary phlebitis, occasioned by coagulation of the blood from its absorption of the products of arteritis, but rather to depend on the transmission of the inflammation from one vessel to another.

This arteritis is of very rare occurrence when compared with phlebitis.

It is occasionally an idiopathic affection, but more frequently it appears to be secondary (metastatic), as it occurs after different acute diseases. The causes on which it depends are frequently very obscure, but in some cases it may be referred to traumatic influences. The form of arteritis which is occasioned by operative agencies, such as ligature, torsion, &c., will, by the process of healing by which it is followed, constitute the subject of future remarks.

General arteritis, like general phlebitis, has no existence.

The above remarks apply to the inflammation of the arteries of the aortic system. In the system of the pulmonary artery appearances indicating inflammation of the larger branches are very rare, and probably are merely secondary processes arising from spontaneous coagulation of blood, and resembling secondary phlebitis. The occlusion of these vessels in most cases results in death, before the development of an excessive inflammation in their coats.

The smaller the arterial vessels are, the more dependent are they on the condition of the surrounding tissue. They likewise participate in this inflammatory process, which either penetrates directly into the vessels of their cellular sheath, or affects them indirectly, in consequence of the inflammatory products penetrating through and saturating the delicate, permeable coat of the vessel. Hence arise the occlusion of the arteries of an inflamed parenchyma, and the obliterations arising from inflammation terminating in induration, as we see in the walls of healing cavities of the lungs.

Although there exists no true *chronic arteritis* of the form here indicated, acute arteritis having products of an adhesive nature may persist for a prolonged period; the textural metamorphosis of its products may be effected slowly, and the terminations we have already indicated may be only very gradually brought about; while arteritis with purulent exudation may terminate in protracted ulceration. That which is commonly regarded as chronic arteritis or as one of its sequelæ, is not originally or essentially inflammation, although it constitutes one of the most frequent and most important diseases of the arteries, as we shall presently have occasion to show.

There is, however, a *chronic inflammation of the arteries, manifested as inflammation of the cellular sheath of the arteries (which consists of a layer of elastic, and a layer of cellular tissue), to which its products are limited, and which merely exerts a secondary disturbing action on the normal relation of the inner arterial coats of arteries, viz., the circular fibrous, and the true lining membrane.* This constitutes a very frequent, and, at the same time, a highly important phenomenon in arteries of large calibre, as, for instance, the trunk of the aorta and its branches.

It is occasionally a *primary*, but more frequently a *secondary* disease, and as such constantly accompanies the morbid deposition on the inner coat of the vessels and its metamorphoses. Its anatomical appearances are in general those of chronic inflammation of the cellular tissue, as, for instance, unusual vascularity of the cellular sheath, with dilatation of the injected vessels, and, corresponding to the degree of its injection, a more or less uniform, saturated coloration, varying from a dark red to a purple, while the cellular sheath is infiltrated with a grayish, or grayish-red, watery or adhesive and gelatinous fluid.

This disease *terminates* in hypertrophy, thickening, and condensation of the cellular sheath, which is converted into a tough, apparently lardaceo-fibrous, and callous, white stratum, varying in thickness from 3 to 6 lines: (Sclerosis.)

It *induces* paralysis of the diseased tissue, more especially of the elastic layer of the vessel, and consequently dilatation, which appears, according to its degree, either diffused, local, or partial. The circular fibrous coat, which is loosened in texture by the dilatation of the vessel, exhibits a morbid brittleness in cases of callous condensation of the cellular sheath, is stratified in appearance, and is of a dirty yellowish, faded color, which indicates a tendency to spontaneous lacerations, owing very probably to the deranged nutrition of the coat of the vessel. Dilatation of the vessel moreover induces the excessive formation of an anomalous inner coat, and its further consequences.

This inflammatory process and the modes of its termination that we have already indicated, are accompanied by two different conditions of the cellular sheath.

In *the one case*, the vascularized, infiltrated, cellular coat of the artery, together with the elastic layer, admits of being detached with unusual readiness from the circular fibrous coat—a condition which may degenerate into spontaneous detachment, and give rise to spontaneous laceration of the lining membrane (dissecting aneurism.)

In *the other* and more commonly observed case, the callous and thickened cellular sheath has coalesced with the circular fibrous coat. This most commonly occurs in dilated aneurismal arteries.

Special reference must be made to *the inflammation of the cellular sheath at the origin of the two arterial trunks, which depends on pericarditis.* It would appear from the milk-spots and adhesions found at this point, that inflammation of this portion of the pericardium occurs very frequently, either partially or associated with general pericarditis. By affecting the subserous cellular substance, it also implicates the cellular coat of these arterial trunks. This inflammatory condition very frequently extends beyond the period of the acute pericarditis, and in the form of chronic inflammation of the cellular sheath gives rise to dilatation, more particularly of the aorta.

In reference to the pulmonary artery, this chronic inflammation sometimes attacks the trunk and its two branches, and it is commonly present in an inferior degree associated with a similar condition of the cellular sheath of the aorta.

b. Ulcerous Processes or destructive ulcerations occur under different conditions in the arteries. They are somewhat frequent, considering the very striking integrity exhibited by the arteries in the midst of extensive abscesses. They invariably originate in the cellular sheath or its vicinity, and in no case do we meet with an ulcerous process either in or upon the inner coat of the vessel. The so-called atheromatous process, which is frequently regarded as ulceration of the inner coat of the artery, is not of an ulcerous character.

To this class belong:

1. The already described suppuration of an artery (see page 95) resulting from arteritis producing purulent exudation—the suppuration arising from arteritis occurring after the application of a ligature, and which will be considered in a future page.

2. Small arterial vessels, together with the capillaries, frequently suppurate when there is suppuration of the different tissues, and we then generally find them in a state of obturation (see p. 196). In less frequent cases, a very violent suppuration of a low character affects vessels in which there is no occlusion, and occasions hemorrhage into the abscess.

3. Ulcerous destruction of the larger arteries in the form of circumscribed corrosion and perforation of the arterial wall from surrounding and contiguous abscesses, constitutes a very important and remarkable phenomenon. The wall of the artery is so much destroyed at a circumscribed spot, that it generally presents a round or oval opening, surrounded either by a smooth, as if cut, edge, or a jagged and fringed margin, and contracted in some instances towards the interior in a funnel-like shape; it is attached by means of this aperture to the abscess, or to the base of the ulcer. This form of ulcerous destruction is the origin of many very dangerous external and internal hemorrhages. Among the most important of these we may instance ulcerous openings of the larger arteries in many different parts of the body. We have ourselves frequently observed perforation of the femoral artery from a suppurating syphilitic bubo, and Hasse noticed such a perforation of the vertebral artery of the right side from an ichorous abscess arising from syphilitic caries of the cervical vertebræ; and to these examples we may add the opening of different arteries on the base of perforating ulcers of the stomach, and the opening of branches of the pulmonary artery by tuberculous caverns.

These ulcerous processes, as we may sometimes notice in the ramification of the pulmonary artery in the walls of tuberculous cavities of the lungs, also give rise to a laceration of the artery. This is owing to the removal of the surrounding protecting parenchyma, and to a loosening and softening of the coats of the vessel, resulting from imbibition of the ulcerous fluid. In some cases this is preceded by a lateral dilatation of the artery towards the cavern.

c. Excessive Deposition of the Lining Membrane of the Vessels.—We rank with the above anomalies a process which, although it does not originally exhibit a diseased textural condition of the arterial coats, at all events results in such, and moreover stands in a near relation to chronic inflammation of the cellular sheath of the artery, the latter being either associated with it, or in rarer instances the indirect cause of its origin, in consequence of previous dilatation of the vessel. It further constitutes the basis of aneurismal formations and of numerous spontaneous obliterations. It is the most frequent form of disease affecting the arteries, and is on that account of the greatest importance. *It consists in an excessive formation and deposition of the lining membrane of the artery derived from the mass of the blood, and at the same time constitutes hypertrophy of this membrane.*—We purpose devoting the following remarks to the consideration of this subject, in which we will endeavor briefly to notice all its most important bearings.

In a highly developed form of this affection, we find the inner surface of a large artery, as the aorta, covered with a foreign substance spread over it at separate points, or in large patches, and forming a stratum varying in thickness, by which the inner surface of the vessel is commonly rendered uneven. This substance is in some places either grayish, grayish-white, faded, and translucent, or in others milky-white, opaque and similar to coagulated albumen; in some rare instances it is colored by the imbibition of hæmatin over various extents of surface. Its free surface is at the same time smooth and shining, or dull and as it were wrinkled. It is soft, moist, and succulent in the translucent parts, and dense, dry, tough, and elastic in the more opaque portions, resembling a cartilage or fibro-cartilage, with which it is usually compared, and for which it is still occasionally mistaken. In the latter condition it adheres internally to the circular fibrous coat.

This substance admits of being split into lamellæ, and drawn away in the form of strata. If this is done at those spots where the deposition forms isolated plates or islands, we discover that one or more of the lamellæ thus drawn away, generally the innermost (superficial) ones, terminate beyond the limits of the plates in a delicate membrane, which is prolonged to the contiguous, and apparently normal lining membrane of the vessel.

The *thickness* and *extent* of this deposition correspond to the degree of the anomalous condition. It varies in thickness from a quarter of a line to two lines and upwards; and extends in extreme cases over the whole trunk and main branches of the aorta, implicating the entire arterial system.

The deposition is generally the thickest directly over the division of a trunk, or at the bifurcation of a vessel. At these points the deposit is frequently so thick, that the mouths of the divergent vessels are much contracted, and even wholly closed.—The spot at the trunk of the aorta, which next to those we have already mentioned, deserves a special reference as a common locality for this deposition, is the lower wall of the aortic arch adjoining the left bronchus. This deposition, which is in itself highly interesting, is rendered still more so when it undergoes ossification. For as we shall have occasion to show, the osseous concretion gives rise here to an angular curvature, and a consequent contraction of the tube of the aorta.

We find on close examination of the deposit, that it has nothing in common with cartilage or fibro-cartilage, with which it is ordinarily compared and even confounded (*cartilaginescence of the arteries*), and that it actually consists of structures analogous to the layers which constitute the lining membrane of the vessel (the epithelium, fenestrated membrane, and longitudinal fibrous coat).

The circular fibrous coat is found, when compared with the other arterial coats, to be soft, brittle, cleft, and of a faded, dirty brownish color. The cellular sheath exhibits considerable vascularity and puffiness, or is in a state of sclerosis.

It will be seen from this description of the appearances observed in the more highly developed stages of the disease, that our attention should, on the one hand, be directed to its incipient stages, and on the other to its further progress.

At its commencement, this deposition cannot be detected without a previous familiarity with its appearance. It is then a delicate, soft, succulent membrane, exhibiting a vitreous transparency, and appears in some cases, where it is thrown into small folds by the preponderating contraction of the circular fibrous coat, to be exceedingly thin, and covered with white dots or stripes. The circular fibrous coat is normal, when not altered in consequence of pre-existing inflammation of the cellular sheath.

The deposition continuously increases in thickness by the addition of new strata, and thus gradually passes from the condition of transparency and succulence, characteristic of recent formations, to that state in which it appears opaque, resembling coagulated albumen, and finally presents a ligamentous appearance, having a dull, wrinkled surface.

Before we enter upon the consideration of the metamorphoses which further occur in this deposit, it will be necessary to direct attention to some points which, although of extraordinary interest, have hitherto been wholly overlooked.

On attentively examining the inner surface of a highly diseased artery, we perceive that the *deposit is interspersed with openings or foramina, varying in size from a pin's head to that of a poppy-seed.* These openings occasionally attract attention by a small drop of blood oozing from them on pressure. In some cases these openings are very numerous, whilst in others it is difficult to detect them. They might, at first sight, be mistaken for the contracted mouths of vessels; but the error of this view is readily made apparent by a closer examination, and by the circumstance that they occur at spots where no such vessels are given off, as, for instance, on the ascending arch of the aorta.

These openings lead to canals, which penetrate to various depths in the deposit, where they either terminate without changing their form, or again divide, and turning, with their branches, in an oblique direction, enter the circular fibrous coat, where they finally ramify. They constitute a system of canals to convey the blood into the deposit and the cleft circular fibrous membrane, which is filled by the blood of the diseased vesel, and may frequently be seen through the deposit.

As far as we are aware, these canals are not connected with the vascular system of the cellular coat, and do not anastomose with its vessels, although they penetrate as far as its elastic layer.

The manner in which these openings and canals originate is a question of the greatest interest. They are undoubtedly the result of partial resorption in the deposit, by means of which openings are formed, which enlarge into canals by coming in contact with each other in the different strata of the deposit. Their mode of origin is very probably similar to that of the apertures of the fenestrated or striated arterial coat, and is closely connected with it; it is very likely, also, the same process which Stilling observed in the thrombus of tied arteries, and which we have also noticed in a fibrinous coagulum in the heart (in the so-called polypus of the heart), where it rendered the coagula porous, and caused them to acquire a cavernous structure, erroneously regarded as a condition of vascularity.

This *channelling* of the deposit undoubtedly constitutes the basis of that degeneration of the arterial walls which Lobstein considers under the head of *softening of the arteries* (artério-malacie). The wall of the artery, in some few cases, degenerates into a spongy tissue, resembling the corpus cavernosum, or occasionally into the form of a tumor, from which, when it is cut, blood pours forth from an innumerable quantity of openings, as from a sponge.

The metamorphoses through which the above described deposit passes, after it has become completely opaque, are the so-called *atheromatous process*, and *ossification* of the arteries.

1. *The atheromatous process* consists in the metamorphosis (disintegration) of the deposit into a pulpy mass, compared by the French to a purée of peas, consisting of a large number of crystals of cholesterin, fatty globules, and of molecules exhibiting various degrees of consistence, from coarseness to extreme fineness, and consisting of albumen and calcareous salts.

The metamorphosis begins with a finely punctuated opacity and decoloration of the deposit, and it is not limited to any definite duration, occurring sometimes at an early stage, and at other times at a more advanced period, although, as has been already observed, generally when the deposit has become opaque. It, moreover, commonly begins in the deeper, older strata of the deposit, and advances from thence towards the surface. It usually affects a space varying in circumference from the size of a lentil to that of a crown or a shilling piece. There is, at the same time, an increase of volume, and a swelling of the deposit; the uninjured lamellæ rising above the surface towards the interior in proportion to the depth to which the process has affected the deposit, and then frequently exhibiting a perceptible fluctuation.

After this process has penetrated to the innermost layers, or when they have burst above the pulpy mass, and been torn asunder by the force of the blood pressing into the cavity, the mass itself appears uncovered on the inner surface of the artery, and in contact with the blood, in which case fibrinous vegetations of different forms are deposited on the fringed margins, after the occurrence of the bursting or rent.

The pulpy mass, both immediately after it has been laid bare and also subsequently, is taken up in different quantities into the blood, although another and the more consistent part of it is infiltrated by the blood, and permeated by its fibrin, and is thus rendered firmer, and, at the same time, colored by hæmatin, in various degrees of intensity, being first of a dark red, then of a dirty brown or purplish, and lastly, of a yeast-like color. In addition to these discolorations, the mass acquires a very peculiar appearance, when its surface is covered with large crystals and accumulations of cholesterin, for it then looks as if it were interspersed with spangles, or silver-like and shining scales.

These spots are even at the present day regarded as *ulcers—fungous ulcers of the arteries.* But the atheromatous process presents no essential analogy with an ulcerous process, nor is the deposit itself an inflammatory product. We discover no trace of an ulcerous product in the atheromatous mass, and its admixture with the blood is not characterized by any marked subsequent symptoms, as we learn from the experience of hundreds of cases.

The atheromatous mass is very often gradually thickened, and converted into a moist, soft, plaster-like substance; and finally appears in the form of a coarsely granular stalactitic calcareous concretion.

This loss of substance is occasionally replaced by a fresh deposit, when the atheromatous mass has either been wholly, or for the most part, taken up into the blood, in which case these spots remain below the level of the inner surface of the vessel, and thus acquire a cicatrix-like and wrinkled appearance, in consequence of the amount of shrivelling of the callous cellular sheath. They also very often acquire a slate-gray or greenish-gray, or black color from the hæmatin by which the tissue is saturated, and which remains on the margins and on the base. They are regarded as *cicatrices of the supposed ulcers of the arteries.*

2. *Ossification is the second form of metamorphosis of the deposit.* This includes the well-known ossification of the arteries. It presents many essential points in common with the atheromatous process, and occurs only in a deposit of comparatively extensive thickness, beginning in the lowest strata, where it is first manifested by the pale turbidity of the deposit, which has then become opaque.

When the process of ossification has been completed, and the metamorphosis has extended throughout the whole thickness of the deposit, the osseous concretion lies exposed. Its form is that of a concavo-convex plate, having a tolerably smooth, even, and concave inner surface, and a rough, nodularly uneven, convex external surface, with irregular and jagged margins. The bony plate is bored through at the point where a branch of the ossified artery is given off, unless the mouth has already been previously closed by the deposit. In arteries of large calibre, as the femoral arteries, the concretions present the form of rings which enclose more or less of the circumference of the vessel.

The *number* and *size* of these concretions are subject to great variations; in some cases they only appear at detached points, whilst in others, the artery appears to be converted into a more or less solid osseous tube.

In large arteries, the exposed bony plates are often partially detached by the current of the blood, when they remain in the vessel at different angles of inclination. Their rough margins readily become the seat of fibrinous vegetations.

This form of arterial ossification exhibits a yellow color, and, in general, considerable density and hardness of texture. It is deficient in the delicately lamellated structure of bone, and has no medullary canals or bone-corpuscles. (See Miescher, Valentin.)

The seat of arterial ossification is the lining membrane of the vessel, which is itself produced in anomalous excess. The bony plate does not, according to the general view, remain stationary between the inner and the circular fibrous coat; nor does it press these layers asunder, and induce atrophy in the former of the two by pressure, but it is developed in the parenchyma, and in the numerous thick superimposed strata composing the recently formed inner coat of the vessel. It at length becomes exposed in the artery, in consequence of the final ossification of the innermost lamellæ, and not simply from their atrophy.

Besides these, other concretions are occasionally observed in the arteries. Thus, for instance, granular or stalactitic calcareous masses are occasionally found upon the inner surface, or the raised margin of these bony plates. These are either thickened, cretified, atheromatous substances (see p. 202), or cretified fibrinous vegetations.

According to our view, although in opposition to that of many good pathologists, these two processes or metamorphoses very commonly coexist; but, it must be admitted, without any marked preponderance in either of them.

Having given the above description of this deposit and its metamorphoses, and having observed that all the different conditions of this deposition very frequently exist in the same vessel, we purpose, in the following remarks, to consider *the relation simultaneously exhibited by the other coats of the arteries.*

1. At the commencement of the deposition, and till it attains some degree of thickness, there is no perceptible alteration in the *circular fibrous coat.* But such alterations become the more apparent in proportion to the increasing thickness of the deposit; for this coat gradually acquires a dirty yellowish color, its texture becomes looser, and it admits, with uncommon readiness, of being separated into fibres and layers; at the same time it loses its elasticity, and yields to the pressure of the column of the blood, whence dilatation of the vessel supervenes to a degree that corresponds to the extent of inflammation already set up in its cellular sheath. It loses its power of resistance, and becomes thinner in consequence of this dilatation.

In the more highly developed stages of this deposit, and when the atheromatous and ossifying processes have become fully established, and even made considerable progress, the circular fibrous coat presents a dirty brown, yeast-like color, and is soft, lacerable, and cleft. As the dilatation of the vessel increases, the fibres gradually separate, and the deposit sinks into the interstices thus produced, where it comes in contact and finally coalesces with the cellular sheath, which has in the mean time been converted into a callous tissue.

According to our observations, this disease of the circular fibrous coat depends on the development of fat—fatty degeneration, by which, analogously with the process observed in the so-called fatty metamorphosis of the muscular tissue, its peculiar ramifying fibres and its elasticity are destroyed.

This coat is directly implicated in the atheromatous process, which extends to it from the deposit, and destroys it.

2. *The cellular sheath of the vessel, in the majority of cases, is found to be in a state of chronic inflammation*—that is to say, in a state of vascularity, redness, infiltration, and puffiness, or has been converted, in consequence of this process, into a layer of white, very dense, callous

tissue of considerable thickness, coalescing with the circular fibrous coat, or with the deposit within its interstices. The following points are of the greatest importance in reference to this condition of the cellular sheath:

a. The intensity of this condition bears no relation whatever to the degree of the deposition, since, in the higher stages of this process, it is occasionally, and in the less developed stages very frequently absent, whilst it never exists in the incipient form of the deposit.

b. This condition must, therefore, be of a *secondary* character, and associated with a certain stage of the deposit. This fact does not, however, exclude the possibility of the converse relation, for as we shall have occasion to show, a primary and substantive chronic inflammation of the arterial sheath may give rise to a local deposit, in consequence of dilatation of the vessel.

The deposit is either *local*, limited to one or more spots of the vessel, or it extends over a large portion of an artery, or over a separate part or the whole of the arterial system. In the former case it depends on local dilatations of the vessel, and on the slowness or partial stagnation of the current of the blood; in the latter, the controlling influence must be a general state of disease, which we would designate as a *constitutional* condition.

The deposit, when appearing as a constitutional disease, occurs almost exclusively in the *arteries*, and only in the *aortic system*. This agrees with the consecutive anomalies, especially the occurrence of ossification of the vessels, aneurismal formation, and obliteration.

Very little relative importance can be attached to any scales purporting to give the frequency of the occurrence of this constitutional affection in the different portions of the aortic system, for whenever the disease appears especially developed in any definite part, the rest of the system—as, for instance, the aortic trunk—will also be implicated.

The trunk of the aorta is most frequently the seat of the disease; and here we find that the ascending aorta, and the arch, are most commonly affected, next the abdominal, and lastly the thoracic portion.

Next in order follow the splenic, the femoral, the internal iliac, the coronary arteries of the heart, the trunks of the arteries of the brain—that is to say, the carotids within the cranium and the vertebral arteries, with their branches,—the uterine, the brachial and subclavian, the spermatic, the common carotid, and the hypogastric arteries.

It is worthy of notice, that certain arteries are only very rarely, and in exceptional cases, subject to even a subordinate degree of this disease; among these we may reckon the mesenteric arteries, and yet more, the cœliac, the gastric, the hepatic, and the epiploic.

This scale corresponds generally to the frequency of the occurrence of metamorphoses in the deposit, such as its ossification, as well as aneurismal formations.

We have not been able to determine, with the requisite accuracy, whether there actually exists such a symmetrical occurrence of the conditions already described (viz.: crude deposition, ossification, and the atheromatous process) in the corresponding arteries of the two sides of the body, as Bizot maintains that he has observed, and regarding which he has established a law; and indeed our views of the constitutional character of the disease prevent our attaching any great importance to the subject.

This disease is of very rare occurrence in the *pulmonary artery and its branches;* but if it be present here, it is always likewise considerably developed in the aortic sytem.

This affection scarcely ever occurs as a constitutional one in the *veins*, for here it always exists as a secondary phenomenon, depending on a sluggishness of the current of the blood, produced by ordinary causes. (See Diseases of the Veins.) On the other hand, the veins are frequently affected by a morbid formation, which, although it may not be purely constitutional, yet presents a very remarkable analogy with the disease of the arteries under consideration: we refer to the so-called *phlebolites* or *vein-stones.* It is, moreover, worthy of special notice, that the deposit in the veins commonly attains a high degree of development when arterial blood makes its way into them. (Varicose Aneurism.)

Sex exercises no special influence on the occurrence of the deposit and its different consecutive conditions; but Hasse appears, on the whole, to be correct in his view, when he states that disease of the abdominal aorta is more frequent, and more highly developed, in women than in men.

Age, on the other hand, gives rise to important differences; but although the disease is most frequent between the fortieth and the sixtieth year, the assertion that it increases in frequency in proportion to the age, and that it occurs in advanced life almost as a normal condition, is not well grounded; for although it may undoubtedly date in many aged persons from a comparatively early period of life, there are many others in whom it is entirely absent. Old age presents, indeed, a mechanical disposition to this affection, from the dilatation of the arteries common to that period. Before the above-mentioned age, the disease is undoubtedly more rare, although frequent even between the thirtieth and fortieth year. Before that period it is very much rarer; and when it occurs prior to the age of twenty years, it is mostly only a local disease, depending on congenital or early acquired anomalies of the trunks of the vessels and of the heart. This observation refers especially to its occurrence during the periods of puberty and childhood.

If, after considering the above remarks, we proceed to the question—In what consists the nature of the disease? we gather the following facts from our examination of all the important points bearing on the subject:

1. *The deposit cannot be regarded as the product* (*exudation*) *of an inflammation of the arteries.* The chronic inflammation of the cellular sheath of the diseased vessel is almost always a secondary consecutive appearance which associates itself with the already established deposit.

2. *The deposit is an endogenous product derived from the blood, and for the most part from the fibrin of the arterial blood.*

3. *Its formation demonstrates the pre-existence of a peculiar crasis of the blood*, which is intrinsically *arterial*, although at the present time we are wholly ignorant of the character of the peculiarity on which

this depends. We must regard the old dogmatic view, which sought the cause of the affection in arthritis, as an opinion deficient in proof.

4. *In proportion to the extent of the disease of the arteries, so much the less likely is it to be combined with tuberculosis;* and this disease undoubtedly is in part the cause of that immunity against tuberculosis which we constantly notice in large aneurisms of the trunk of the aorta. The grounds of this relation are not known; but it is not wholly improbable that this immunity may arise from a similarity between the process of deposition (which occurs in the form of separation of fibrin), and the tuberculous process, by exhausting the arterial character and the materials of the blood. On the other hand, we very frequently observe an *excessive production of fat associated with the deposition and ossification in the arteries.* This abnormal formation occurs—independently of the fatty degeneration of the circular fibrous coat, and of the atheromatous process,—more especially in the neighborhood of the ossified arteries with atrophy of the muscular tissue, in the vicinity of aneurisms, and, in addition, as excessive accumulations of fat in the blood, of cholesterin in gall-stones, &c.

5. *The deposit and its metamorphoses present numerous, highly important analogies, that have hitherto been wholly neglected.* For the sake of brevity we will here notice only the most important; viz.: the deposit also occurring under certain conditions in the veins, the phlebolites (which we will consider under Diseases of the Veins), the capsules investing different fibrinous coagula in the vascular system, and causing them to adhere to the walls of the vessels and of the heart, and the metamorphosis which these fibrinous coagula undergo within the vascular system, and which may even affect fibrinous coagula externally to that system.

The *effects* produced by this disease in its reaction on the whole organism are still unknown. In respect to the vessel itself, the disease gives rise to different forms of dilatation, with contraction of its branches and complete closure of their mouths, constituting a highly important, although little known, secondary condition. When arteries of lesser calibre have been ossified, and the deposit continues to exist, they finally become closed and obliterated. It is, moreover, probable that the capillary arteries at the seat of the deposit become diseased, in consequence of the diminution and cessation of nutrition arising from the obstruction and arrest of their permeability. Either may, moreover, give origin to the formation of spontaneous gangrene—the so-called dry gangrene—mummification of the tissues. Finally, this disease is often found to terminate in spontaneous laceration of the large arteries, and especially of the small trunks.

It still remains for us to add the following remarks to the observations already made in reference to the diseases of the valves of the heart at p. 174. The valves of the aorta exhibit a thickening and an adhesion to the wall of the vessel, and appear fused together with consecutive shrivelling, malformation, and ossification. We have already remarked, in the same page, that this disease which generally forms the basis of that insufficiency of the aortic valves which is slowly and almost imperceptibly developed in advanced life, is not of endocarditic origin, but

ends upon an excessive formation of a tissue analogous to the inner
t of the vessel, and deposited from the blood upon these valves. It
ommonly associated with a diffused deposition in the trunk of the
ta and a dilatation of the latter with aneurismal formation.

!. *Adventitious products.*—A very few of these forms occur in the
cular system generally, and especially in the arteries.

.. Among the productions of *fibroid tissue* we may include sclerosis
the cellular sheath in consequence of its chronic inflammation, and
haps also that metamorphosis of the deposit in which it becomes con-
ted, in many cases, into such a tissue:

!. An *anomalous production of bone* occurs in the following forms:

:. *The well-known ossification of the arteries—a metamorphosis of
deposit*—to which we have already sufficiently alluded. The fre-
ncy of its occurrence in the different portions of the system, corre-
nds with that of the morbid deposition in the arteries on which it de-
ds. A similar relation exists in reference to the periods of life and
sex in which it most frequently occurs.

'. *Chalky, mortar-like concretions*, formed by the thickening of the
eromatous mass, or by the metamorphoses of the fibrinous vegetations
osited on the above-named bony plates, &c.

. *Ossification of the occluding plug*, which owes its origin to inflam-
ion with exudation on the inner coat of the vessel, after the previous
version of the plug into a fibroid string.

}. The numerous forms in which the *anomalous production of fat*
urs are extremely important. The cholesterin which is contained in
so-called atheroma of the arteries, is the only fatty product that has
n hitherto noticed (Gluge, Gulliver). Yet the fatty degeneration of
circular fibrous coat (see p. 203), which may be compared to stea-
s of the muscles, is, in our opinion, more important in its results,
lst the excessive formation of fat that is combined with ossification
the arteries, present numerous points of interest, both on its own
ount and in consequence of many analogous conditions (see p. 206).

l. *Cysts* are of very rare occurrence in the arteries, and probably
y appear in the cellular sheath and the neighboring tissue. We have
er yet observed a case of this nature. Corvisart attempted to build
a theory of the formation of aneurism on two cases which he had
erved, but his views were long since refuted, and never met with sup-
t.

). *Tuberculosis* neither occurs here nor in any part of the vascular
tem. The deposit does indeed, as we saw at p. 206, give indications
n analogous process, which gives interest to the fact that this disease
s not occur in the venous system as a constitutional one.

). The larger arteries with thick walls steadily resist the invasion of
ncerous Degeneration, and in this respect present a striking contrast
the veins. Whilst the veins traversing a cancerous tumor exhibit
cerous degeneration of their walls, and are often completely filled up
cancerous excrescences, the arteries are found to be undestroyed.
ile the spontaneous coagulation of the blood in the arteries is rare,
find that the tissue developed from it is still more rarely of a cancer-
nature. Velpeau and others have observed the very rare occur-

rence of an obstruction of the aorta and iliac arteries by a plug of a cancerous nature, in an individual exhibiting a general cancerous dyscrasia. The rarity of cancer of this form—*primary cancer of the arteries*—is worthy of notice, when contrasted with its more frequent occurrence in the veins.

§ 4. *Anomalies of Calibre.*

A. *Dilatation of the Arteries* (*Aneurism*).

As we purpose limiting ourselves, in the following pages, to the consideration of the conditions of actual dilatation of the artery, we will postpone to another chapter the consideration of false, varicose, and dissecting aneurisms, as subjects which do not appropriately belong to the present place.

After simply referring to the development of the arteries in reference to their calibre and the thickness of their walls, as observed in organs having become hypertrophied (in the gravid uterus and in morbid augmentations of size in the most different organs), we will pass on to dilatation of the arteries based on disease of the arterial coats.

It seems, however, especially necessary to notice, in the first place, the conditions under which dilatation of an artery is established. These are as follow:

1. Some dilatations arise without any visible alteration of texture *in consequence of a loss of elasticity and contractility of the coats of the artery*, that is to say, of the circular fibrous coat and of the elastic sheath, or in consequence of *a mechanically induced continuous and excessive filling of an artery or even of one entire section of the arterial system.* Such dilatations are very commonly observed, particularly in advanced life, in the trunk of the aorta, and especially in the ascending arch, *in consequence of the first above-named causes.* If, as we think, not improbable, Cloquet's two cases of *circoid aneurism* (or *arterial varix*), belong to the class in which the aneurism is not the result of extensive chronic inflammation or of deposition, such a dilatation might be extended over the larger portion, or even the whole of the arterial system. In such dilatations the walls of the arteries are thinner, softer, and more yielding, and the circular fibrous coat is paler than usual. The dilatation, especially at the trunk of the aorta, excepting where it exhibits a more or less strongly marked protrusion of the convex portion at the ascending aorta, presents a regularly cylindrical form: in the so-called *circoid aneurism*, however, it appears so far irregular that it preponderates in one or other portion of the surface, and thus gives rise to the interlaced twisted course of the artery (which seems as if it were coiled round an imaginary axis), and to the bulgings occurring at certain points, as well as to the lengthening of the diseased vessel.

Dilatations arising from mechanical obstructions are observed in the aorta; but more especially in the pulmonary artery and its branches, in consequence of the various diseases of the parenchyma of the lungs which impede the normal injection of the capillaries, in cases of stenosis of the left side of the heart, &c.

2. *Chronic Inflammation of the cellular sheath of the arteries* gives rise, as has been already remarked, to dilatation of the vessel, in consequence of its paralyzing the elastic coat. This is especially observable in the trunk of the aorta, and it may, moreover, have laid the foundation of some or other of the recorded cases of *cirsoid aneurism.* Dilatations of the point of origin of the trunks of the pulmonary artery and the aorta, depending upon inflammation of the cellular sheath, combined with pericarditis, deserve notice on account of their locality (see p. 122). These dilatations are generally of a cylindrical form.

The forms of dilatation based upon the two causes above described, constitute (more particularly in reference to the arterial wall enclosing the dilatation) *true Aneurism* (*Aneurisma verum*), which has been distinguished by the designation *Arteriectasis* from those aneurisms which are regarded as depending for their origin on a more essentially anatomical disturbance.

3. *Most forms of dilatation commonly included under Aneurism* (*spontaneous aneurism*), *and at the same time the most important of all, are owing, as we have already shown* (p. 199), *to the deposition of a tissue, analogous to the lining membrane of the vessel, derived from the blood, and occurring upon the inner surface of the artery, and to consecutive disease of the circular fibrous coat and the cellular sheath.* Attempts based on an examination of the mode of construction of their walls, have been made to separate these dilatations into *true* and *false or spurious aneurisms;* or in accordance with a principle at variance with Scarpa's view of the cellular sheath of the vessel, into *true* and *mixed aneurism,* the latter being subdivided into *external mixed* and *internal mixed* or *hernial aneurism.* This class, moreover, comprises the dilatations which Cruveilhier and others, distrustful of the results of anatomical examination, divided merely in accordance with their external forms, into *diffuse, fusiform, cylindroid* and *sacciform aneurisms.* The last class were divided by Cruveilhier into "*A. sous l'aspect d'ampoules*" with subdivisions into "*A. périphériques, semipériphériques,*" and "*à bosselures,*" and "*A. sous l'aspect de poches à collet.*"

These dilatations considered under the common term of aneurism—spontaneous aneurism—will form the subject of the following remarks.

We must, however, begin by observing that a classification of aneurisms, simply based on the anatomical conditions of the coats of the artery, can only have reference to *gradual disturbances,* and cannot, therefore, afford a representation of essentially different and well-defined species, where the grounds that give rise to the formation of aneurism are the same. Although, indeed, a division derived from external form may have some practical utility, it cannot afford a sound classification, inasmuch as it has no reference to the anatomical disturbance on which the form depends, nor can it separate well-defined species, owing to the numerous transitions of form which they undergo.—We do not purpose giving any special description of the arrangements made by different observers of these forms, since the references made to them in the appropriate parts of our work will be sufficient for their correct apprehension.

Spontaneous Aneurism appears in its simplest type as a diffused dilatation of a vessel towards all points of its surface in a *cylindrical form,* or when, as is usual, it gradually decreases towards both its extremities, till they assume the normal calibre, it is *fusiform* (the *A. diffusum cylindroideum* of some writers, Cruveilhier's *A. sous l'aspect d'ampoules périphériques*).

Where the diseased condition of the coats of the vessel affects a more or less sharply-defined and considerable portion of the surface, the vessel dilates at that point in the form of an originally shallow pouch, which is gradually converted into a sac, flattened at its edges, where it is in contact with the interior of the artery, constituting *saccular Aneurism*, Cruveilhier's *A. sous l'aspect d'ampoules semipériphériques*. The same is the case when, in diffused disease of the vessel, the morbid condition preponderates at any one point of the surface; the vessel being then generally dilated, but more especially at this point, whereas in the former case it is dilated into the form of a sac with ill-defined margins, and flattened at the spot where it branches off from the vessel.

It will be readily understood that a cylindrical or fusiform aneurism may very commonly be converted into a saccular aneurism, in consequence of the *preponderance* of disease at one or other point of its walls; or, even in the absence of disease, this change of form may arise at any point especially exposed to the force of the blood-wave, as for instance at the convexity of the ascending aorta.

These aneurisms are not only remarkable for their frequency, but more especially for the *extraordinary size* which they very commonly attain. The most important among them are those in which the saccular expansion affecting a portion of the periphery of the vessel is so situated that the wall opposite to the sac retains its normal form and direction. These approach to the pedicled aneurisms.

Saccular Aneurisms are commonly of a round form, although they are occasionally oval or conical in shape, even from their commencement; more frequently the form loses its roundness in consequence of excessive disease of the coats of the vessel at different points, when the dilatation preponderates more or less in one direction.

Finally, *secondary pouches in the form of roundish or conical elevations, or protuberances—Cruveilhier's A. sous l'aspect d'ampoules à bosselures*—not uncommonly occur in the aneurisms already described (viz., in cylindroid, fusiform, and sacciform aneurisms), when the disease of the coats of the vessel preponderates at several generally inconsiderable, although somewhat sharply-defined points. These protuberances may in their turn give rise to excrescences of a secondary form (*a tertiary aneurismal formation*), which project in various ways over each other, giving the vessel the appearance of an irregularly lobulated sac.

When a cylindrical or fusiform dilatation attacks a vessel irregularly over a considerable portion of its surface, and preponderates at different alternate points of the periphery, causing the vessel to extend in a longitudinal direction, by which its course becomes twisted, and as it were distorted round its axis, we again have the *form of aneurism termed cirsoid.*

A study of the development of the above forms yields the following facts in relation to *the construction of the walls of these aneurisms.*

When the dilatation has not exceeded a certain degree, the walls of all these aneurisms consist of the whole of the diseased coats, and hence constitute *true aneurism.* The internal stratum is formed by the deposition in its different conditions of opacity, fibroid metamorphosis, atheromatous process or ossification, and in a state of actual channelling. It represents the lining membrane of the vessel. Next follows the decolorized and cleft circular fibrous coat in the act of being metamorphosed into fat; and, lastly, the elastic and cellular coat—the cellular sheath—whose fibres are entwined with one another, and with those of the circular fibrous coat in a hyperæmic and vascular condition, and which exhibits either a bluish red coloration, or pallor and sclerosis.

When the dilatation is very considerable, the tissue of the circular fibrous coat is found to be much separated, while the stratum seen through it is thinner than usual. When the dilatation has extended beyond a certain limit, the fibres of the circular fibrous coat are not only completely separated, and even wanting at several inconsiderable portions of the aneurism, but this coat gradually disappears at the borders of the highest elevation of the fusiform or saccular aneurism, and at the margins of the superposed secondary pouches; the wall of the aneurism consisting here only of the diseased lining membrane of the vessel and of the cellular sheath. At these pouches the dilatation constitutes the so-called *internal mixed* or *hernial aneurism*, which we purpose considering more in detail.

There is a form of aneurism presenting considerable interest in many points of view, which Cruveilhier has designated *A. sous l'aspect de poches à collet, and which may be termed pedicled aneurism.* It differs very decidedly in its marked external form from the saccular variety already described, and is separated from it by many stages of transition.

This aneurism resembles a round sac, which is in general attached to the diseased vessel by means of a neck-like base or contraction. This base corresponds to an opening into the vessel, which is equal in circumference to the contraction, and is either round or oval in form, and surrounded by a projecting margin. It constitutes the channel of communication between the vessel and the aneurism.

No form of aneurism presents such striking variations in magnitude as this; since, without reference to the calibre of the vessel, it may exhibit every possible size, from that of a pea to that of the fist, or even of a man's head. The most common size is from that of a walnut to that of a middling-sized apple. These aneurisms are, moreover, distinguished by their tendency to burst when still of very inconsiderable dimensions, as for instance, when they are not larger than a pea or a bean.

An examination of the walls of these aneurisms yields the following facts:

1. *In most cases, the wall of the sac at its base near the opening into the artery, consists of the wall of the artery that has been everted by the aneurism and of all the diseased coats of the artery, whilst the circular margin surrounding the opening is formed by a duplication, as it were, of the entire wall of the vessel.* At different distances from this point, however, the circular fibrous coat, after having become gradually thinner, entirely ceases, and the wall of the aneurism then consists almost wholly of the deposition (the inner coat of the vessel) and the cellular

sheath. The margins of the opening are smooth and covered by the deposit; the aperture is roundish.—These appearances present themselves in very small as well as in large aneurisms of this nature. *This form of aneurism almost invariably occurs as a secondary formation, being seated on a cylindroid or fusiform aneurism.*

2. *In some few cases the circular fibrous coat terminates sharply at the margin of the aperture in the artery.* The aperture is generally irregular and angularly contracted, *whilst the wall of the saccular pouch above it consists of the cellular sheath and of a deposit*, which projects from the contiguous inner surface of the vessel over the margin of the circular fibrous coat in the form of bridge-like plates and strings, and adheres loosely to the cellular sheath in the cavity of the expanded portion of the vessel. This appearance is observed only in small aneurisms before they exceed the size of a bean or a hazel-nut, and they then commonly prove fatal by bursting. *They usually occur as primary aneurisms, and in general in arteries that are only slightly and locally diseased.* The aperture in the circular fibrous coat is obviously the result of loss of substance.

3. In cases of similarly rare occurrence, we meet with a sharply-defined bulging of the artery, filled with the atheromatous mass resulting from the disintegration of the deposit and the circular fibrous coat. *The wall is here composed of the cellular sheath.*

On considering the above relations, we arrive at the following conclusions in reference to the origin and form of these aneurisms.

Although nothing positive can be determined in reference to the question, whether the appearances considered under the first head are the result of the further development of the appearances included under the second head, it is however very probable that such is not the case. The aneurism considered under the first head appears to be the result of excessive disease of the coats of the artery at a circumscribed spot. The artery bulges, and its wall then bends at the margin of the diseased tissue towards the tube of the vessel, with which it forms, as it were, a duplicature of the wall of the artery. Finally, the circular fibrous coat gradually separates at the top of the bulging, when the deposit, consisting of the diseased lining membrane of the vessel, is brought in contact with the cellular sheath with which it coalesces within this cavity. The further enlargement is now especially exhibited at this point, until the wall of the aneurism finally consists, for the most part, merely of the deposit and the cellular sheath.

This is undoubtedly the form that has been named *external mixed aneurism* (Scarpa's *Aneurysma spurium*), and is believed by some observers to consist merely of the cellular sheath of the artery, in consequence of their having regarded its investment as unessential and as a recent formation, and from their inability to trace the whole of the layers passing into the aneurism. Since we are disposed from our view of the subject to regard the investment as originally formed by the diseased lining coat of the vessel (although certainly in a condition of expansion and attenuation) which has coalesced with the cellular sheath in the aperture formed in the circular fibrous coat, we may regard this aneurism as the same which has been named by other observers *hernial aneurism.*

The appearances considered under the second head, have undoubtedly been developed from those noticed under the third head; at all events we are unable to discover in what manner this loss of substance has taken place within the wall of the vessel, unless by the atheromatous process described under the third head. We find that the deposit and the circular fibrous coat are affected throughout and destroyed. The atheromatous mass is gradually lost by being absorbed into the blood, and hence the bulging at this point consists of the cellular sheath.—This aneurism, when considered in accordance with the above-described mode of origin, is, strictly speaking, an *external mixed aneurism;* but in this form it probably never constitutes the subject of anatomical investigation. Thus, for instance, an inner coat of the vessel is produced in the form of a recent deposit, which renders it difficult, or indeed almost impossible, to recognize and distinguish this aneurism from others, especially when it has existed for a prolonged period. This aneurism does not appear, however, to be of long continuance, but generally bursts while of inconsiderable size. It is, moreover, of very rare occurrence when compared with other aneurisms, and cannot therefore have been the solo means of giving rise to the theory in reference to external mixed aneurism, or of originating the opinion of the frequency of its occurrence.

We take the present opportunity of answering the question, *Whether a rent in both the inner coats of the artery can give rise to the formation of an aneurism of this class?* The belief in this mode of origin has met with almost universal accordance, although, as far as we know, the correctness of the opinion has never been proved by any one. The cavity in the neck or pedicle of these last-named aneurisms has commonly been regarded as a fissure. Yet, as far as we are aware, no such rent has ever been detected, nor have we ever found that a fissure in the inner coats of the artery afforded a basis for the formation of an aneurism. (Compare Laceration of the Arteries.)

We believe that the above observations comprise all the most important points in reference to the form of spontaneous aneurisms and the construction of their walls, however much they may seem at variance with a sharply-defined classification of aneurisms in accordance with any fixed principle. By way of completing our observations, we will only remark, that the deposit covering the inner surface of the last described aneurism which is attached by a neck, is also found to be affected by the different conditions of opacity, atheromatous disintegration, and ossification.

There still remain several other appearances in the aneurism which demand our attention.

1. *The cavity of the aneurism very frequently contains fibrinous coagula*, which usually form very distinctly stratified masses. The external and older layers consist of a whitish fibrinous substance, generally more or less deprived of color, and of a faded appearance. They are dense, compact, tough like leather, and dry. The inner layers constantly become looser, more moist, and colored, until at length the innermost—those of most recent formation—resemble a recent coagulum of blood. The fibrinous layers frequently exhibit many other conditions of great interest. Thus, for instance, the external, denser layers become, in some cases, converted into a whitish, callous texture, which coalesces

with the wall of the aneurism, and very considerably strengthens it. In some cases they present an ossification similar to that which appears in the fibroid exudations, as, for instance, on the serous membranes; while, at other times, they are observed to be softening into a yeast-like yellow or whitish pulp, or a cream-like fluid.

As an important phenomenon which is often presented, we may notice that a recently formed layer of the lining coat of the vessel is inserted at different points between the strata of the fibrinous coagulum, giving the whole mass the appearance of being invested with such a membrane, which is then prolonged into the deposit investing the interior of the vessel. We here discover the means employed for restoring and maintaining the continuity of the vessel by closing the cavity of the aneurism with a new layer of the lining membrane of the vessel.

The fibrinous layers in the aneurism fulfil, therefore, no other purpose than that of assisting mechanically to maintain the coagulation of the blood and of its fibrin. They are not the product of an inflammatory process in the wall of the aneurism, nor do they exhibit the character of a malignant growth.

These coagula are not present in every aneurism. As a general rule it may be assumed that, without reference to the size of the aneurism, they will be present in large quantities, in proportion to its distance from the axis of the blood-current, and to the smallness of the communication between the cavity and the calibre in the vessel, when compared with the size of the aneurism. Hence we see the reason why fibrinous coagula are so much more readily and extensively deposited in aneurism of the pedicled form, which presents these two requirements in the most highly developed degree, and why their formation becomes the less easy when the aneurism differs from the above and approximates to the spindle-shaped or cylindrical variety. In these and saccular aneurisms, the formation of the coagula depends only on the extent to which the wall of the pouch recedes from the axis of the blood-current. It is, moreover, natural that fibrinous coagula should occur in larger quantity in large than in small aneurisms of the same form.

These fibrinous coagula derive importance from the obstruction they oppose to the rapid increase of the aneurism and to its early bursting, and in consequence of their causing a wasting of the aneurism, and thus inducing its spontaneous healing.

2. *These aneurisms* differ very considerably *in their dimensions*, as has been already observed, varying from the size of a pea or bean to that of a man's head, and thus occasionally filling up the greater part of one of the large cavities of the body. In general, the largest aneurisms occur on the large arteries, more especially on the trunk of the aorta; but there is no invariable proportion observed between the size of the aneurism and the calibre of the vessel, for aneurisms fully equal in size to those which occur on the trunk of the aorta, are occasionally met with in vessels of inferior calibre, as, for instance, the femoral and popliteal arteries.

Large aneurisms experience a very extensive alteration in the construction of their walls, to which sufficient attention has not been paid. Until the aneurism has acquired a certain degree of enlargement, it retains *its primary wall*, whose composition we have already described; but when the aneurism exceeds these limits, and the wall is no longer equal to the expansion, its place becomes supplied by adventitious tissues and structures, either over the whole extent, or at more or less sharply-defined spots, corresponding to the direction of the increase in volume. These are the structures with which the aneurism is in contact during its increase, and with which it gradually coalesces. This circumstance explains the reason why aneurisms, which only increase very slowly in volume, and therefore are only gradually brought in contact with structures able to compensate for the loss of substance of their walls, may attain so great a size, whilst those aneurisms which are rapidly formed and enlarged, and are, therefore, not brought in contact with many of these structures, speedily burst. These adventitious products are accumulations of cellular substance, serous and fibrous membranes, muscular expansions, &c., together with parenchyma, as, for instance, that of the lungs.

We must distinguish between the manner in which aneurisms of great size lose their primary wall, and the loss arising from the result of detritus—the absorption occasioned by pressure where the aneurism is in contact with bone. Thus we find, that where aneurisms, even of very inconsiderable dimensions, are in contact with bone, the aneurismal wall, together with the periosteum, is partly destroyed and the bone exposed.

3. We have already considered all the essential points in reference to the *form of these aneurisms*. It will be evident that the vicinity of resisting structures may, in various ways, modify the form of the aneurism during its growth. Thus aneurisms on the descending aorta occasionally assume a bilobar form posteriorly, in consequence of the resistance offered by the vertebral column, which causes it to separate into two sacs lying on either side.

4. We find great diversity in the *number* of aneurisms which may be simultaneously present. In some cases, several aneurisms are present together, either on different arteries or in close vicinity to each other on the same artery, so that the tube of the vessel exhibits a row of adjoining and even confluent aneurisms. Large aortic aneurisms are usually isolated, which may be explained, at least in part, by the weakening of the mechanical force through the carrying off of a large quantity of blood towards the aneurismal sac.

5. The greatest interest and the most important results arise from *the relations exhibited by the branches passing from an artery affected with aneurism; they consist in narrowing or entire closing and displacement of the mouths, and the consequent atrophy of the vessel.* These results are produced by various and often intimately connected means.

a. A highly developed degree of deposition (see p. 206), very commonly gives rise to the important conditions of *contraction, and, finally, complete closure of the mouths of vessels opening into the artery affected with aneurism.* It more especially affects the mouths of small vessels branching off from the diseased trunk, either at a right or an obtuse angle, as, for instance, the mouths of the intercostal arteries, and of other vessels branching off from the diseased thoracic aorta; although it also not unfrequently implicates the mouths of vessels of larger calibre, as, for instance, those of the carotids, the subclavians, &c.

b. Secondly, the mouths of the branches of the vessels are also rendered insufficient and are displaced by means of the ***fibrinous coagula deposited on the wall of the aneurism.*** They have commonly been already contracted by the deposition, or have been rendered insufficient by means of the fissure-like opening, which we shall shortly notice. This imperviousness of the mouths is more especially limited to those vessels which branch off from the diseased trunk, either at right or obtuse angles.

In consequence of the displacement and closure of the mouth, the blood which reaches the branch of the vessel through the collateral circulation coagulates, and the vessel is then obliterated from above the plug to the point where the next branch is given off.

c. Thirdly, the branches passing off from a diseased trunk are rendered insufficient *by the round form of the mouth being contracted and altered into a cleft-like opening*, which is frequently rendered still more impervious by the projection of a valve-like margin which inclines backwards in the direction of the heart. This is more especially found to occur in the branches of the arch of the aorta, when the latter is the seat of large saccular dilatations.

d. Finally, there is a mode of obliteration that occurs in the vessels branching off from an aneurism, either independently, or complicated with the above-described forms. *This mode of obliteration is the result of inflammation with exudation upon the inner surface of the vessel, and of the subsequent coagulation of the blood.* It appears only in vessels having thin walls, and which are, therefore, liable to this form of inflammation.

The effect of the aneurism on neighboring parts is to displace and press upon them, in proportion to their inability to offer any resistance to this pressure. By these means the functions of the injured organs are either partially or entirely obstructed. Thus aneurisms of considerable size may variously contract the space of the cavities of the body, and either diminish the apertures of different passages, such as the trachea, the bronchial tubes, the œsophagus, the arteries and veins, &c., or compress them so powerfully as to render them entirely impervious.

Pressure gives rise in different structures to various alterations which are proportional to the degree of pressure and the capacity for resistance presented by the tissue. Moderate pressure generally occasions inflammation in the contiguous structures, which gives rise to condensation and thickening—increase of bulk. When the pressure exceeds a certain limit, it results in atrophy. Both of these results are, however, frequently combined, being found simultaneously present in different parts of the tissue; thus, for instance, the parts in the immediate vicinity of the aneurism may be atrophied, whilst the more remote tissue exhibits a new formation of cellular substance and of fibroid tissue. We very frequently observe that bones which have been exposed to the action of an aneurism exhibit atrophy (detritus), whilst various osseous formations —osteophytes—occur at detached points surrounding the aneurism, and even sclerosis may be present in the contiguous bony layers.

Yielding membranous expansions in part give way to strong pressure, while their fibres admit of being separated; and in part they become gradually atrophied like cellular tissue, serous and fibrous membranes, muscular coats, &c. Large masses of muscle become pale and thin, and even wholly disappear.

Highly vascular and nervous structures, such as the external investment and the mucous membranes, have their texture so much loosened by inflammation, that they readily tear; or where this is not the case, they become gangrenous.

Parenchymatous structures waste away in consequence of the exudation produced by inflammation, and finally become atrophied.

Vessels are obliterated either in consequence of coalescence, induced simply by perfect compression, or in consequence of inflammation, that is to say, by means of adhesion to a coagulum of blood produced by the inflammatory process.

Nerves undergo atrophy through pressure and tension.

Rigid structures become atrophied in proportion to their deficiency of elasticity. Detritus of the bones is therefore very commonly induced by aneurism, whilst cartilage and fibro-cartilage, as, for instance, the intervertebral cartilages, are longer able to resist this action. This detritus is most frequently observed in the bodies of the vertebræ, in the ribs and the sternum, the clavicle, and also in the scapula in aortic aneurisms, and is often present in so highly developed a degree, that these bones are entirely destroyed, and the osseous wall of the thorax perforated. The vertebral canal has even been seen opened.

The process of resorption induced by the deposition and pressure of an aneurism on the bones, destroys not only the osseous substance itself, but, sooner or later, the aneurismal wall also, which becomes fused as it were with the periosteum and the other fibrous structures that usually invest the bones. The bone is then either very commonly laid bare, or is only covered by a layer of the deposition investing the aneurism, or by the fibrinous coagula in the aneurismal sac. The exposed vertebral column thus very frequently constitutes a portion of the aneurismal wall. In aortic aneurisms which perforate the anterior or lateral wall of the thorax, the roughened and nodular extremities of the ribs, the clavicle, and the sternum, are almost entirely denuded on their inner surface, and project into the sac of the aneurism.

The effects of the aneurism are diffused beyond its own immediate locality to distant organs, and even over the whole organism. These effects are as varied in their nature as the influences from which they arise; but in general they occur more rapidly, are more violent, and are more extensively diffused in proportion to the size of the aneurism, its relation to a main artery, and its vicinity to the heart.

The pressure on the nerves and their tension occasion variously developed symptoms of neuralgia and paralysis.

The pressure of the aneurism gives rise to a varicose condition of the veins below the aneurism, venosity, cyanosis, dropsy, and inflammations, which frequently terminate in gangrene.

Large aneurisms on the trunk of the aorta have a tendency to produce active dilatation of the heart, and this tendency is the more marked in proportion to their vicinity of that organ. They give rise to this disease either in association with insufficiency of the aortic valves, which is, however, generally the case, or independently of this affection. They also induce general venosity, diffused, as it were, from this point, as from a centre.

The pressure on the arteries, and the occlusion resulting from it in the region of the aneurism, may possibly be unattended by injurious results, in consequence of the establishment of a collateral circulation.

The stasis and coagulation of a considerable quantity of blood within a large aneurism, have the effect of withdrawing so large a quantity from the organism, as to occasion symptoms of anæmia, tabes, a watery condition of the blood, general dropsy, and cachexia. The pressure of the aneurism on parenchymatous structures, and the obstruction of their functions, contribute without doubt to the presence of cachexia, and to the development of its special character.

The following must be noticed in reference to *the modes of termination of aneurism:*

Aneurism very commonly terminates fatally.

This fatal termination is very frequently induced by the results already mentioned, amongst which we may specially place diffused inflammations terminating in gangrene, dropsy of the cavities of the body, hyperæmia and acute œdema, more especially of the lungs, cachexia, and general marasmus.

Spontaneous opening or laceration, rupture and extravasation of blood from the rent constitute a very frequent, always extremely unfavorable, and indeed very often rapidly fatal termination of aneurism. We would direct attention to the following particulars in reference to this subject.

The tendency to spontaneous opening does not bear a direct relation to the size of the aneurism, for we find that small aneurisms burst more frequently than larger ones.

The direction in which the aneurism opens, and in which the blood emerges, varies considerably. Aneurisms in the limbs open into the surrounding cellular tissue, in consequence of which a large quantity of blood is extravasated into the intermuscular, subcutaneous cellular tissue, below and between the aponeuroses, the muscular sheaths, &c. Aneurisms of the trunk, and of some of the branches of the aorta, as, for instance, the splenic, open into the large cavities of the body, as the peritoneal sac, one or other of the pleural sacs, or the pericardium, occasioning hemorrhage into the corresponding cavity and the sub-serous cellular substance. Aneurisms of the cerebral arteries open in a similar manner into the sac of the Arachnoid, and into the tissue of the Pia Mater.

Aneurisms very frequently open into canals, as the trachea, the bronchial tubes and their large branches, and œsophagus, and more rarely into the intestinal canal and the cavities of the urinary passages. They however very commonly open into other bloodvessels, either arteries or veins, and even into the cavities of the heart, more especially the auricles. Such openings very frequently occur in aortic aneurisms into the trunk of the pulmonary artery and its branches, and into the ascending or descending Vena cava.

Aneurisms that are imbedded in parenchymatous structures do not often open; the hemorrhage here takes place into the parenchyma, and after the latter has been extensively displaced or perforated in the form of a canal, the blood flows freely into the adjacent serous cavity.

We have seen one instance in which an aneurism of the aorta opened into a tuberculous pulmonary cavity having healed, consolidated walls.

Finally, aneurisms may sooner or later penetrate to the general investments, and open externally.

The manner in which aneurisms open is not the same in all.

Aneurisms which project into a serous cavity burst at that part which, having coalesced with the serous membrane, and become extremely thin from a deficiency of the surrounding tissue adapted to strengthen and protect it, offers the slightest degree of resistance in consequence of the excessive attenuation of its walls. The opening generally occurs at one of the most saccular portions of this wall, and is either in the form of a fissure, or more frequently of a roundish aperture having a fringed margin. The latter appearance induced Hasse to believe that the opening was preceded by a pre-existing and self-induced process of softening, but we have never been able to detect its presence in the numerous observations in which we have been engaged.

In those cases in which the aneurism bursts through the walls of the canals on which it is seated, and opens into their cavities, the process is more complicated. Thus aneurisms open into the trachea, the bronchus, and the œsophagus, when the fibro-cartilaginous and muscular elements, together with the adhering wall of the aneurism, are destroyed by detritus, in consequence of the mucous membrane becoming the seat of inflammation, and tearing in that condition with the aneurismal wall. In other cases, as for instance at the œsophagus, *a gangrenous eschar* is developed in the mucous membrane over the encroaching aneurism, and, by extending over the whole of the aneurismal wall, usually gives rise to extensive opening of the aneurism.

The opening of the aneurism into the cavity of the neighboring blood-vessels is brought about in various ways. In some cases the aneurismal wall coalesces with the cellular sheath of the adjacent artery in such a manner as to deprive the circular fibrous coat of the latter of its proper support. As the aneurism exerts a stronger impulse on the vessel, the cellular sheath becomes completely separated from the artery, and, consequently, the aneurism and its circular fibrous coat at length burst. The rent is in general large, and presents an angular form in this coat of the artery; it is usually complicated with detachment of the cellular sheath over varying extents of surface from the fissure. (See Dissecting Aneurism.) In other cases, the cellular sheath of an adjacent artery coalesces not only with the aneurismal wall, but also with the circular fibrous membrane of the affected artery, in consequence of a very chronic process of inflammation, and the slow development of the aneurism. The circular fibrous coat directly coalescing with the aneurism is thus rendered thinner, whilst its fibres separate from one another, and at last wholly disappear at different points. At the point which corresponds with the most marked protrusion of the aneurism into the artery, the aneurism bursts together with the layers of the lining membrane of the artery coalescing with its wall. The rent, as in aneurisms that open towards a serous cavity, is small, fissure-like, or resembles a roundish hole.—The opening of aneurisms into a contiguous vein is effected in the same manner. (*Spontaneous varicose aneurism.*)

We occasionally find in aneurisms imbedded in parenchymatous organs and cellular accumulations, that there is an acute inflammatory process, which hinders the development of a protecting and strengthening callus, and by predisposing the tissue to softening and laceration above the pulsating spot, occasions laceration. In other cases the tissues are separated by the pressure, without the concurrence of any such inflammatory process, and thus give rise to the rupture of the aneurism.

When an aneurism opens outwards on the surface of the body the process depends, as in aneurisms that open into the mucous canals, on a high degree of inflammation in the true skin, occasioning a separation or laceration of the tissue, or on a gangrenous eschar implicating the general investments.

Such openings are very often rapidly formed and *single*, although occasionally we observe *several small perforations*, so that there is at first only a gradual and recurring oozing of blood, until the opening acquires a very considerable size.

Finally, the wall of a cylindrical, spindle-formed, or saccular (true) aneurism, frequently exhibits perforations which are owing to a laceration and detachment of the diseased inner coats from the cellular sheath of the vessel. But this is a subject to which we shall revert when we pass to the consideration of the spontaneous lacerations of arteries.

The above forms of aneurismal ruptures in ordinary cases produce death by hemorrhage, externally, or into one of the large serous cavities, or into the trachea, the alimentary canal, &c. When the aneurism opens into other vessels, such, for instance, as the arteries in the vicinity of the heart, or into any of the cavities of the heart, the result is in general speedy death, in consequence of the obstruction in the circulation. There are, however, exceptions to this rule; and we find that in some instances, small perforations of the latter kind may exist for a prolonged time without causing death, in which case the aperture through which the communication is maintained, acquires a smoothed, healed appearance, from its margin being invested with a recently formed lining membrane. This is more especially the case when the aneurism opens into a vein, and thus constitutes a basis for the formation of a so-called *spontaneous varicose aneurism* (Thurnam).

However unfavorable the ordinary termination of an aneurism may be, instances are occasionally observed in which *the disease takes a more favorable turn, and nature brings about a spontaneous cure of the aneurism.* This result is effected in many different ways, which have been especially considered by Hodgson.

1. *The aneurism may compress the artery on which it is seated in such a manner, either above or below, that it gradually becomes impervious, and is then obliterated with the aneurism.* We attempt to imitate this healing process artificially, by passing a ligature either above or below the aneurism. Such a result can only affect saccular aneurisms, and such as are attached by a neck.

2. *The aneurism may be completely filled with fibrinous coagula above which a deposit is formed, which represents the lining coat of the vessel,*

and stops the communication between the cavity of the aneurism and the tube of the vessel. Aneurisms attached by means of a neck, and having only a narrow passage of communication, present the most favorable conditions for this mode of termination. We even observe, in some rare cases, that where saccular or spindle-shaped aneurisms have been completely filled with fibrinous coagula, new formations continue to be deposited upon the former, until at length the whole diseased vessel becomes obstructed. Decrease in the general quantity of the blood and a diminution of the heart's action must be regarded as the most favoring influences.

In both these conditions the aneurism shrivels and contracts over the coagula, either in the form of a fibroid capsule or of a spindle-shaped cylindrical roll, and is then atrophied.

3. *In aneurisms in the extremities the gangrenous process to which they give rise attacks the aneurism itself, and by exciting arteritis, causes the artery to be stopped up by a coagulum.* The aneurismal sac is thrown off and removed, and the artery obliterated at various parts. In the same manner, abscesses and inflammatory foci in the vicinity of an aneurism may occasion arteritis, accompanied by occlusion, and subsequent obliteration of the artery, by which the aneurismal sac is destroyed, and removed by suppuration.

Spontaneous aneurism generally occurs with the same proportional frequency in the two arterial systems, and in the different portions of the aortic system as the disease of the coats of the vessel on which they are based (see p. 204). The relative scales of frequency established by different observers are indeed tolerably accurate; but still many of the results which have been given are incorrect; thus, for instance, the assumption of the great frequency of aneurism of the popliteal artery, is undoubtedly so far incorrect that it includes in the same class with spontaneous aneurisms of the lower limbs, which are certainly not of rare occurrence, many others which very probably were of traumatic origin.

In general, aneurisms are incomparably more frequent in the larger than in the smaller arteries, and their occurrence on the trunk of the aorta is characterized by remarkable frequency.

Aneurisms are, on the contrary, very rarely observed in the pulmonary arterial system, where, as far as we know, they are limited to the trunk. Fusiform and saccular dilatations do certainly sometimes occur in the ramifications of the pulmonary artery, within the parenchyma of the lung, as we have observed near tuberculous caverns; but as they originate from entirely different causes, they do not belong to this class (compare p. 198).

Aneurism of the trunk of the pulmonary artery is scarcely ever present, unless there is at the same time aneurism of the aorta, or, at all events, a tendency to that disease.

Although it may be said in reference to the *sex* most frequently affected that there is a preponderance in men, it is by no means so considerable as is usually supposed. The *age* at which aneurisms are most common is between the 30th and 60th year; they are of much rarer occurrence between the 20th and 30th year, and must be regarded as extremely unfrequent, and as exceptional cases, when they are present before the age of 20 years. We must, however, exclude from this calculation all the aneurisms of traumatic origin which have hitherto not been separated with sufficient care.

It has long been supposed that aneurisms are *based on a special aneurismal diathesis*, in consequence of the frequency with which they have been observed to appear spontaneously and independently of external influences, and from the fact that several occur simultaneously or in quick succession to each other in the same individual. Thus incidental and individual cases have led to the idea that these predisposing conditions were to be sought in gout, syphilis, or mercurial cachexia; and this opinion was supposed to derive support from the more frequent occurrence of aneurisms in men, as well as from the period of life at which they are most commonly observed. It was conjectured that this diathesis gave origin to the diseased condition of the texture of the coats of the vessel, and to their loss of elasticity and their softening and brittleness.

We have sufficiently considered the anatomical bearings of the disease affecting the coat of the vessel on which the formation of spontaneous aneurism depends (see p. 199), and have drawn attention *to an anomaly of the blood-crasis, which may give origin to an aneurism* (see p. 206). Nothing positive is known in reference to this blood-crasis; but the concurrence of the above-named diseases with aneurism appears to us to be purely accidental, nor do we think that such individual cases afford sufficient scientific grounds for the connection that has been supposed to exist between these diseases and the aneurism.

It is a very important fact *that spontaneous aneurism never exists in combination with tuberculosis.* This immunity is based on the following grounds:

a. The diseased condition of the coats of the vessel on which aneurisms depend, constitutes a cause of immunity against tuberculosis (see p. 206).

b. Large aneurisms of the aorta give rise to consecutive disease of the heart in the form of dilatation, with a readiness proportional to their vicinity to the heart. It is, therefore, in consequence of the venosity and cyanosis occasioned by the latter disease, that aneurisms of the aorta afford a decided immunity against tuberculosis.

Aneurisms, as we have already seen, have nothing in common with cancer.

It still remains for us specially to notice several particular forms of aneurism.

Aneurism of the Aorta.—The aorta is more frequently the seat of aneurism than any other vessel, and the parts most commonly affected by aneurismal formations, are the ascending aorta, and the arch.

The aneurisms most common on the trunk of the aorta are the cylindrical and spindle-shaped aneurism, the saccular form affecting only one side of the vessel, the pedicled, and even the cirsoid aneurism, which is occasionally observed along the entire length of the tube of the aorta.

Saccular expansions very frequently occur in the *ascending aorta* at the sinuses, and especially at those two which correspond to the convex wall of this portion of the aorta. These aneurisms very frequently burst at an early stage into the cavity of the pericardium, and occasionally into the right auricle.—Pericarditis in some instances gives rise to the rupture of aneurisms projecting into the cavity of the pericardium.

Aneurisms are incomparably more frequent on the convex than on the concave side of the ascending aorta.

The same is the case in reference to aneurism of the arch of the aorta.

On the *aorta descendens*, aneurisms within the thorax appear most frequently to proceed from the posterior wall and the sides of the vessel, so that they very commonly implicate the vertebral column and the adjacent thoracic wall.

Aneurisms of the abdominal aorta are usually spindle-shaped and saccular, and are most frequently developed from the anterior and lateral portions.

An extensive series of observations has afforded the following particulars in reference to the remarkable peculiarities presented by these aneurisms.

1. Those aneurisms which arise from the convexity of the ascending aorta, and from the anterior and upper wall of the arch of the aorta in general attain a very considerable size, inclining in such a direction that they touch the right half of the sternum, the costal cartilages, and the ribs of the right side from the first to the fifth or sixth, or even extend to the sterno-clavicular articulation and the right clavicle, finally destroying the parts by detritus, and coming to view externally in the corresponding region of the thorax. It is important to remember that such is their course, from which there are very few exceptions, because an aortic aneurism occurring at the sterno-clavicular articulation and at the right clavicle is very commonly mistaken by the bedside for a subclavian aneurism, which is in general erroneously supposed to be of great frequency.

2. Aneurisms, proceeding from the concavity of the ascending aorta, extend in the direction of the pulmonary artery, or are seated in front of it, towards the wall of the left auricle, and open into one or other of these parts.

Those aneurisms which proceed from the concavity and posterior portion of the arch of the aorta, abut upon the trachea and the bronchi, and in general open into them at an early period, and long before they have attained any considerable volume.

3. Aneurisms of the thoracic aorta commonly first implicate the vertebral column at the part corresponding to the above-described points of origin, and destroy it to various extents, and in rare cases, to such a degree that they come in contact with the *dura mater* of the spinal chord, and even burst into the canal. They moreover diffuse themselves over the posterior wall of the left side of the thorax, and occasionally open freely into its cavity, or, in some rare cases, so completely destroy the thoracic wall as to come to view externally on the back. They very often implicate the left bronchus, make their way into the pulmonary parenchyma, and open into it, or into one of the larger bronchial tubes within the lung.—When they occur on the right side of the vessel, they are situated in the mediastinum and on the œsophagus into which they open.

4. In the very rare cases in which aneurisms of the abdominal aorta

burst, their contents are usually effused into the cavity of the peritoneal sac.

On Dilatations of the Ductus Botalli.—The dilatations which in rare cases are observed in the Ductus arteriosus, in every period of life, from the earliest infancy, are simple, and not dependent upon any alteration of texture in the coats of the vessel. They are occasioned by a deficient involution of the duct after birth.

If we except that degree of patency of the Ductus arteriosus, in which, in consequence of a uniformly deficient involution (closure), it remains similar in calibre to a branch of the pulmonary artery in newborn infants, and forms a very secondary cylindrical vessel, we find the following different forms of dilatations, which admit of being referred to an unequally deficient closure of its mouths.

1. In *one case*, when the occlusion of both mouths has once commenced, the process goes on more slowly in one—probably the aortic mouth, whose calibre continues permeable after the other mouth has become considerably contracted. Blood now collects here, dilating the vessel, and gradually coagulating within it, forming a spindle-shaped or round, spherical capsule (aneurism), after which this mouth also is finally closed. This anomaly is unquestionably devoid of importance, and does not lead to any secondary consequences, as the coagulum, and the coats of the vessel over it, gradually shrivel together.

2. In *other cases* the Ductus arteriosus is found to present a funnel-like dilatation from the aorta, and the opening into the pulmonary artery is then surrounded by a torn and fringed margin. That this anomaly is not a true patency,—a persistence of the Ductus Botalli in its original form and significance,—is made evident by the above-mentioned relation of the duct, and more especially of its mouths; by its violent reopening from the aorta, towards the pulmonary artery, as indicated by the character of the mouth; by the occlusion of the Foramen ovale, which is observed in such cases; and the existence of a current opposed to the fœtal circulation, and inclined from the aorta towards the pulmonary artery.

This condition is owing to the relation of the duct and its openings, that is to say, to the violent reopening of the closed ostium of the pulmonary artery from the dilated aortic portion of the duct; and also to the active dilatation present in these cases in the right side of the heart, which is one of the results of the obstacles produced by the entrance of arterial blood into the current of the pulmonary artery.

On Traumatic Aneurisms.—These are aneurisms which patients refer to some traumatic influence, such as a contusion, shock, or some unusual muscular effort, &c., and which the physician, in the absence of all disease in the coats of the vessel, must regard as having such an origin. Aneurisms of this character especially occur in the arteries of the extremities, and, as we have already observed, they are too commonly included without further inquiry under spontaneous aneurism, when they are undoubtedly of traumatic origin. To this class belong a certain number of aneurisms of the femoral, popliteal, and brachial arteries.

We are here led to inquire what disturbance is set up in the wall of the vessel by the traumatic influence, which can give origin to the formation of the aneurism.

This question is very difficult of solution; for on the one hand, we rarely or never have an opportunity of examining the artery immediately after the accident, while on the other an examination of an aneurism, when already developed, does not afford absolutely valid grounds for judging of the original disturbance. It is, however, very probable that this disturbance may be based upon *some traumatic influence inducing paralysis of the circular fibrous coat at the affected spot; destroying its contractility, and causing a separation of its fibres; and occasioning a partial laceration of the coat, not affecting either the integrity of the lining membrane or of the cellular sheath of the vessel.* We are led to this opinion from a consideration of the following facts:

1. We cannot believe that traumatic aneurism can be produced by spontaneous laceration of the lining and of the circular fibrous coat of the artery, and therefore be owing to the dilatation of the cellular sheath at the spot where the rent occurs. We have never, for instance, seen an aneurism arise from a separation of the continuity of the lining and circular fibrous coats when it appears either as a spontaneous or a mechanically induced rent; but, on the other hand, there is always, in these cases, a more or less violent and extended detachment of the cellular sheath, whether the arterial coats be healthy or diseased, constituting a secondary laceration of the sheath, and effusion of blood over the vessel. (See Dissecting Aneurism.)

2. In consequence of the great toughness and power of resistance of the cellular sheath of the artery, a separation of continuity can only be effected in all the coats of the vessel when the shock or contusion has been such as necessarily to produce extensive and repeated laceration of both coats. The consequence of this would at all events be to produce effusion of blood from the vessel over a considerable extent, giving rise to an evident *false diffused aneurism* which would also be subsequently apparent in the consecutive condition of *false circumscribed aneurism.*

3. The dilatation developed at the affected spot manifests itself originally as a circumscribed and gradually enlarging tumor which is slowly developed, and shows both by its form and construction the probability of the view we have advanced in reference to the disturbance on which traumatic aneurism is based. This dilatation exhibits either the form of a saccular expansion, or of a pedicled aneurism according to the degree of depression of vitality and loosening of continuity produced by external influences in the circular fibrous coat. Its walls principally consist of the lining membrane of the vessel and of the cellular sheath; in the first form we find remains of the circular fibrous coat between the other membranes, while in the second form the fibres of this coat are separated through external agencies; the lining coat of the vessel adhering within the interstices to the cellular sheath, and gradually protruding through it. The attenuation which the lining membrane of the vessel must necessarily undergo, cannot be directly observed, in consequence of the new membrane which has been simultaneously formed in the aneurism. This second form of traumatic aneurism appears therefore to be a *hernial aneurism*, according to the signification we shall attach to it in the following remarks.

On Hernial Aneurism.—The existence of a *hernial aneurism*, or of an *internal mixed aneurism*, has formed the subject of numerous investigations, from the time of Haller to our own day.

We have already become acquainted with aneurisms, which, as we incidentally observed, must be regarded as hernial aneurisms, in consequence of the anatomical disturbance to which they owe their origin, and in consequence also of the construction of their walls. It now remains for us more closely to define the sense in which we are led from experience to admit the existence of a hernial aneurism.

If it be requisite for the establishment of a hernial aneurism that it should exhibit a dilatation of the lining membrane of the vessel in the form of a hernia through an opening in the middle and outer coat of the artery, we must wholly deny the existence of such a form of aneurism.

1. The direct experiments of J. Hunter and E. Home showed that the removal of the external coat of an artery did not give rise to the protrusion of the lining membrane in the form of an aneurism. Whether the external coat alone, or that and the middle one, were both loosened and detached, the result was simply inflammation and cure without any alteration in the calibre of the injured artery.

However limited may be the application to be extended for various reasons to the results of these experiments, they are still highly interesting, and must excite our surprise from the opposition in which they stand to the result we should have been led to expect. We are not astonished merely at the circumstances that, after the removal of the outer and the middle coat, the lining membrane did not protrude, but still more that, considering its slight power of resistance, it did not at once give way. The circumstance that the middle and lining membranes were not lacerated after the removal of the outer coat, is very probably owing to the elastic sheath having been left on the vessel, and not removed with the outer coat.

2. To the results of these experiments we must add those yielded by observations on human arteries.

Detachment of the sheath of an artery, which consists of an elastic and a cellular layer, is not attended, as we learn from observations of the so-called dissecting aneurism, by a saccular expansion of the exposed yellow and lining membranes, but by its immediate laceration, both in those cases in which it is owing to external influences, and those in which it has resulted spontaneously from a morbid process. When, moreover, this occurrence is met with in cases where the middle and lining membranes were observed to be healthy, we are the more led to conclude that it would exist where there is disease of these membranes.

In such ulcerous perforations of the arterial wall from without inwards, as we noticed in the femoral arteries (see p. 198), notwithstanding the probably gradual and stratified separation of the different layers of the tube of the artery, we perceive no trace of aneurismal formation at the affected spot.

There is, on the other hand, a form of aneurism very frequently met with, which, when considered in the following sense, represents *hernial aneurism.*

a. In spontaneous, spindle-shaped, saccular aneurism, the diseased circular fibrous coat gradually yields at spots which vary in number according to the size of the aneurism. The lining membrane of the vessel (the deposition) coalesces in the interstices thus produced with the cellular sheath, and wherever these portions are excessively dilated, the secondary aneurismal formation deposited on a *cylindroid*, *fusiform*, *or sacciform aneurism*, gives rise to aneurisms of a secondary form, commonly known as *hernial aneurism*.

b. Aneurisms that are attached by a neck, and that are composed for the most part merely of lining membrane and cellular sheath, essentially constitute *hernial aneurism*, in as far as they are produced in the same manner as the above-named secondary dilatations.

c. Finally, *traumatic aneurism*, in accordance with the process from which it arises, and which has already been described, is a *hernial aneurism*.

Dubois, Dupuytren, Breschet, and others have undoubtedly taken a similar view of the question, when they maintained the existence of a hernial aneurism. We would, moreover, specially remark, although the circumstance seems sufficiently evident from the foregoing observations, that the inner coat of our hernial aneurism is by no means composed of the original lining membrane of the vessel, but consists almost entirely of newly-deposited strata.

We do not, for obvious reasons, regard the establishment of hernial aneurism as a separate class to be essential, nor do we think it possible, in all cases, to separate it strictly from spontaneous aneurism.

B. *Abnormal Narrowness—Contraction—Obliteration of the Arteries.*

The arterial system presents numerous varieties of irregular narrowness, and, moreover, exhibits many differences in respect to its extent and degree. To this class belong *congenital anomalies*.

1. *A Congenital Abnormal Narrowness of the Aortic System*, which is strikingly apparent in the large arteries, and more especially in the trunk of the aorta. This is found in some cases, in adults, to be contracted, particularly in its descending arch, to the calibre of an iliac or even of a carotid artery. This anomalous condition, which is very generally associated with deficient development of the system, and with a striking thinness and softness of the arterial walls, is often overlooked in childhood, and very commonly does not exhibit any distinct symptoms until the period of puberty, when it manifests itself by insufficiency in the calibre of the artery compared to the quantity of the blood, and by dilatation of the heart, more especially of the left ventricle. It most frequently occurs in females, and is combined with retarded development generally, and more especially with smallness of the sexual organs.

Anomalies of various extent, amounting even to entire occlusion, are occasionally exhibited in the trunk and branches of the pulmonary artery, occurring as congenital conditions, combined with, and depending on obstructions in the interior of the heart.

2. *A Congenital deficient Development of separate Portions of the Arterial System*, more especially in relation to the calibre and elabora-

tion of the coats of the vessels supplying undeveloped, stinted parts and organs of the body.

Acquired Abnormal Narrowness appears under many forms, and frequently attains so great a degree as to present complete occlusion of the artery. All the various contractions, and the atrophies in which they result, may be principally referred to *a simple involution of the artery*, to *contraction and obliteration in consequence of disease of the coats of the vessel*, to *occlusion of the artery*, and to *contraction and obliteration depending on pressure on the artery.*

1. *Contractions and Obliterations in the Form of simple Involution of the Artery.*

To this class belong the following:

a. The contraction and subsequeut atrophy which affect the arteries of organs that are becoming atrophied through accidental or intentional (operative) injuries inflicted on portions of the body which have been previously arrested in their growth.

b. An obliteration which is very similar to the atrophy of the fœtal passages, as for instance, that of the Ductus arteriosus. Such an obliteration of an artery is occasioned by the establishment of a collateral circulation, which is especially induced by a congenital narrowness (obstruction) of the artery in question. The vessel becomes narrower in proportion to the progressive development of this collateral circulation, and is entirely closed when the latter is completed. To this class undoubtedly belong many cases of obliteration of the different arteries, the causes of which have not been sufficiently explained, and most certainly *those cases of obliteration of the aorta at its arch beyond the part where the Ramus brachiocephalicus is given off, corresponding to the depression of the Ductus Botalli.*

These cases, which embrace the majority of the observations made on obliteration of the aorta, have hitherto been unexplained, both in reference to the malformations on which they depend and the process giving rise to final obliteration. They have been repeatedly collected and arranged (Barth, Craigie).

From the interest which attaches to these obliterations of the aorta we are induced to add the following remarks, which are derived from the observations above referred to, as well as from my own experience.

1. An inconsiderable portion of the arch of the aorta, generally at the part already referred to, becomes sooner or later obliterated. The aorta, that is to say its descending portion, is generally abnormally narrow before the establishment of complete obliteration.

2. The aorta before this point, and the branches given off from its arch exhibit considerable dilatation, which extends from these branches over all their ramifications and anastomoses.

3. The heart is in a state of general dilatation, although the left ventricle is the special and original seat of the affection, which also extends to the trunk of the pulmonary artery and its branches.

A careful consideration of all the circumstances leaves little doubt that the following theory is correct.

1. *This anomaly is based upon a deficient formation*, consisting in the permanence of the aorta in that early fœtal condition in which it

constitutes a trunk which merely supplies branches to the head and upper extremities, whilst the pulmonary artery bends round towards the descending aorta in the form of the future Ductus arteriosus, and supplies branches to the rest of the body. The ascending aorta, after giving off its three branches, merges as a thinner vessel into the pulmonary artery. If the branch of the pulmonary artery which bends down to the descending aorta, and represents the Ductus arteriosus, be closed, —which, singularly enough, happens in all cases,—the descending aorta cut off from the pulmonary artery, approaches the ascending aorta so closely as to leave only a very narrow connecting link between them, viz., the thin vessel already described, merging into the pulmonary artery which represents the descending aorta.

2. *This portion of the vessel, from its narrowness, presents the conditions requisite for its obliteration and atrophy.* It becomes narrower, with increasing years, in relation to the ascending aorta and to the quantity of blood passing through that vessel, while, at the same time, not only the ascending aorta, but the branches given off from it are proportionally dilated. This dilatation soon extends over all the ramifications and their anastomoses, as, for instance, those of the internal mammary and the first intercostal with the remaining intercostal and the epigastric arteries. In proportion as the collateral circulation draws the blood more freely from the left side of the heart and from the isthmus between the arch and the descending aorta, the former becomes narrower, and is at length rendered useless, completely closed, and finally atrophied.

The heart is, in all these cases, more or less distinctly affected with active dilatation. This dilatation obviously depends at first on the narrow isthmus between the ascending and descending branches of the aorta, and, after the latter has become atrophied, on the inefficiency of the collateral circulation.

This contraction and closure of the aorta has been observed from the fourteenth to the ninety-second year. It occurs far more frequently in men than in women. There are fifteen or sixteen such cases on record.[1]

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[1] These cases, as already remarked, were collected by Barth (Presse Médicale, 1837), and by Craigie (Edinburgh Med. and Surg. Jour., Oct. 1841). Craigie enumerates ten cases, among which, according to Hasse's statement (Path. Anatomie, Bd. i. p. 91), one of the cases collected by Barth must be wanting. I unfortunately have not Barth's memoir by me at the present moment. He collected nine cases, which include Otto's case (which Hasse must have missed in Barth's collection, as he specially enumerates it, although it is not given in Craigie's list), Römer's case (Oest. Jahrb., Bd. xx. St. 2), and the case observed by Craigie, and described in the above-named memoir. These, together, make twelve cases, to which four others have been recently added—one case observed by M. Aug. Mercer in the year 1838 (which Craigie has overlooked), a case observed by Muriel in 1842, one described by Hamernjk in 1843, and one case, also occurring in 1843, which is preserved in our Pathological Museum.

We think that a more detailed notice of the last-named cases may contribute to the completion and elucidation of Barth's and Craigie's series.

The *thirteenth case*, observed by M. Aug. Mercer (Bulletin de la Société Anatomique de Paris, xiv. année, p. 158):

Contraction, with almost complete Oliteration, of the Thoracic Aorta.—Potier, a shoemaker, aged 38 years, was received into La Charité on the 29th of March, 1838. He had been seized, in the October of the preceding year, with violent bleeding from the nose, which continued for three hours. This occurrence of epistaxis relieved him from attacks of giddi-

Death is generally occasioned by the heart-disease and the anomalies to which it gives rise; in four cases it was owing to rupture twice of the

ness to which he had been previously subject. Towards the month of January he suddenly experienced a sensation of cold and weakness (paralysis) in the right hand, which disabled him from using his knife. This sensation disappeared in about a week. From the 27th of February he had had an occasional cough; but it was not until the 27th of March that blood was expectorated, which increased very considerably on the 28th and 29th.

The patient experienced pain in the region of the apex of the heart, and forwards and backwards at about the same elevation, which prevented him from lying on his side. The beats of the heart were frequent, but without any peculiar sound; but at the summit of the arch of the aorta, a strong bellows-sound, continued into the carotids, was heard in unison with the arterial pulse. The sound was almost equally loud at the lower angle of both scapulæ, where two or three of the intercostal arteries were observed to pulsate with violence. The pulse at the wrist was 140, very large and hard, but otherwise regular. There was no sound along the femoral arteries, which beat so faintly that they could scarcely be felt. An obstruction to the current of blood in the descending thoracic aorta was diagnosed.

On the 31st there was a violent pain at the top of the ninth dorsal vertebra, between the spinal column and the scapula. The symptoms of pleuro-pneumonia increased, notwithstanding energetic treatment; and the patient died on the 9th of April.

Autopsy.—The left pleural sac contained a coagulated and fluid exudation; the lung was in a state nearly approximating to pneumonia in its third stage.

The heart was large, and invested with pseudo-membranous coagula; the aorta, together with the arteries branching off from it, was dilated from its commencement to about a few lines below the origin of the left subclavian. In the middle of the free margin of one of the aortic valves, there was seated a whitish-red, and apparently old coagulum.

About five lines below the point of origin of the left subclavian, the aorta appeared to be almost entirely obliterated. The opening, which would only admit a blunt probe, was closed up with coagulated blood. It was linear in form, and surrounded by a posterior and an anterior lip, the latter of which projected far less than the other, causing the opening to approach nearer to the anterior than the posterior wall of the aorta. The tissues appeared to be normal at the contracted spot.

The Ductus arteriosus was obliterated, terminating in the concave portion of the aorta, about three-fourths of a line above its contraction.

The contraction extended over a very inconsiderable space, and was sharply defined both at its commencement and its termination. Immediately below it, the calibre of the aorta scarcely varied perceptibly from its normal dimensions. The abnormal aorta, and the iliac and femoral arteries, were also only slightly smaller than usual. The pair of intercostal arteries, branching off above the contraction, were 2‴ in diameter; the remainder gradually decreased in calibre to the fourth, which appeared to be normal.

Fourteenth case, described by William Muriel in the seventh volume of "Guy's Hospital Reports" for 1842.

James Bert, a laborer, aged 25 years, of small stature, died on the 27th of July, 1842. Nine years previously, he had suffered from symptoms resembling those of an aneurism of one of the larger vessels of the chest. The symptoms gradually abated under the proper treatment, and, after a few months, he had so far recovered as to be able to work again; and was employed as a farm-servant uninterruptedly till the 20th of June, 1842. On that day, however, on lifting a heavy weight, he sprained himself. This accident gave rise to pain in the back and spasms, which were alleviated by opiates and counter-irritants applied over the spine. He lingered, however, until the 27th of July, when he died in a comatose state, which had been preceded with severe pain in the head.

On a post-mortem examination, the body was found to be somewhat emaciated, the chest deformed by the projection of the sternum, more especially towards the ensiform cartilage, and there was an inclination of the spine in the upper dorsal region towards the right side; the pericardium contained about three ounces of fluid; the heart was somewhat hypertrophied, with some dilatation of the ascending aorta, and of the vessels branching off from the aortic arch. At the point of union of the Ductus arteriosus, the aorta was *extremely contracted and almost obliterated*, whilst the superior intercostal arteries, more especially on the left side, were much dilated. There was no malformation of the heart. Opposite the contracted portion there was a hard tumor, about the size of a hen's egg, which was intimately connected with the aorta and the trachea, and formed by the bronchial glands. The left sides of the bodies of the third, fourth, and fifth dorsal vertebræ were partially destroyed in the region of the tumor; the lungs and the other viscera were healthy; the head and spine were not examined.

ascending aorta, once of the right ventricle, and once of the right auricle. Here, as is generally the case with those heart-diseases which frequently

Fifteenth case, described by Dr. Jos. Hamernjk, of Prague (Oesterr. Wochenschrift, 1843, No. 10).

N. N. Maurer, aged 42 years, who had always enjoyed good health, had been injured eighteen years previously by the upsetting of a carriage, which occasioned contusion and a dislocation of the scapular end of the clavicle. He was seized with pains in the feet and œdema; and stated that, although these symptoms disappeared in a few days, he suffered ever since the accident from palpitation of the heart and headache, which were always removed by spontaneous epistaxis. About ten days before his death, he was attacked with pneumonia of the right side, and died on the 13th of February, 1843.

Dr. Hamernjk saw him two days before death, and found the temperature of body higher than usual, the pulse at the wrist 120, and tolerably large. The pulsations of the heart might be seen and felt between the sixth and seventh ribs, while at the same time the next two upper intercostal spaces sank inwards; the same happened with the first-named intercostal space at the diastole, whilst the two intercostal spaces above bulged out. The resonance in the region of the heart was not strikingly diminished, but the sounds of the heart, and along the greater arterial trunks, were not distinct, excepting perhaps the second sound in the pulmonary artery and the aorta, which were strong and very clear. A clear blowing sound was perceptible over the whole surface of the cardiac region, somewhat after the cystole. This sound was strongest at the left border of the sternum, from whence it diminished in clearness, although it might be heard at a considerable distance (from the dilated internal mammary artery, which was rough). There was pneumonia of nearly the whole of the right lung, of which only the upper part seemed free. A somewhat rough bellows-sound was perceptible in the carotids and the subclavians, as well as in the other large arteries. This sound was rather clear, and loud and protracted at the back, to the left of the vertebral column, at the posterior extremity of the second rib, and might be heard over the whole length of the vertebral column. Bulgings of the compressed and pulsating arteries were to be seen over the whole surface of the back, running in a twisted manner, and more especially diffused on both sides of the vertebral column in the direction of the axilla. There was no œdema.

On opening the body, pneumonia of the right lung was discovered. The heart was somewhat large; the cavity of the left side, however, was small, although its walls were upwards of an inch in thickness; the valves were normal, as was also the pericardium. The above-described rolls of pulsating bulgings along the vertebral column, were the dilated and attenuated branches of the transverse arteries of the neck and scapula, as well as of the subscapular artery.

Dr. Hamernjk himself only saw two separate portions of the body, which he describes:

1. On *the walls of the chest* the two internal mammary arteries were laid open; their calibre was enlarged to the thickness of the little finger; their coats were interspersed at various points, with some few uneven cartilaginous plates, more especially at the upper portion of the vessel.

2. *The portion of the Aorta.*—The arch of the aorta, as far as the left subclavian, was only about an inch and a half in length; it measured 7‴ in diameter, and its walls exhibited their normal thickness and elasticity. The left subclavian artery was 6½‴ in diameter, and was therefore nearly as large as the remaining portion of the arch of the aorta. There were scarcely 2‴ of the subclavian artery remaining in the preparation, and the outer wall was invested with some thin plates of bone, as was also the posterior wall of the descending portion of the aorta. About one inch below the point of origin of the subclavian from the aorta, the latter was suddenly contracted circularly, but more especially at the back, by a deep furrow, so that, with its walls included, it did not exceed 5‴ in diameter when measured from right to left. The contracted portion scarcely measured 4‴ from before backwards, and was therefore somewhat flattened. Above the confined or contracted portion, the aorta was swollen to about the size of a middling-sized hazel-nut, and ossified. At the point of contraction, a transverse wall was observed, having the form of a bi-concave lens, and about 1–1½‴ in thickness, which entirely closed the tube. Below the contracted portion, and about 2‴ deeper, the aorta began suddenly to dilate, and measured 12½‴ in diameter. This dilatation extended over a length of about an inch and a half. The aorta then again assumed its normal diameter; and about 1 inch above the diaphragm it was very slightly dilated. The intercostal arteries of the right side, more especially the second and seventh, were dilated; the former was at least double its normal width. Its walls were thin and collapsed.

The remains of two small shrivelled vessels, with contracted tubes, lay close together on the concave wall of the contracted spot, where each ended in a *cul de sac*. They correcontinue unnoticed for a long time, and do not give rise to disturbances in the system until they have attained a certain limit, the patients continued perfectly well up to a certain period, when the symptoms of heart-disease were either gradually or suddenly manifested.

sponded to the opening of the Ductus Botalli. Dr. Hamernjk was not able, from the restrictions imposed on his use of the preparations, to discover whether this character depended on original division (duplicity) of the arterial passage, or whether it could be regarded as owing to acquired shrinking or puckering. There was no roughness or cartilagescence to be seen below the obliteration.

Dr. Hamernjk is of opinion that these appearances were due to original formation.

Sixteenth case, in our pathologico-anatomical Collection. Dr. Dlauby, who conducted the post-mortem examination, has given me the following particulars:

Harzmann Ignaz, aged 27 years, a day laborer, suffered for some years before his death, from slight erysipelas of the face, during one winter and a succeeding autumn. For more than a twelvemonth he had experienced considerable palpitation of the heart at night, after hard work. During the last three months, this had been frequently associated with cough and expectoration of tough mucus, and with oppressed respiration. This condition grew rapidly worse; and for two months before his death, which occurred in the beginning of March, 1843, he had œdema of the feet.

Autopsy.—The body was of a robust make; there was œdema, more especially of the lower extremities; the abdomen was much distended and fluctuating.

The sinuses of the dura mater were distended; the pia mater, together with the brain, abounded in blood.

The abdominal cavity contained about 20lbs. of clear serum, intermixed with scattered fibrinous flocculi. The liver was not much enlarged; its substance was distinctly separated into a yellow and a dark reddish tissue (nutmeg liver); its peritoneal investment was thickened, and in some spots had a tendinous appearance. The gall-bladder contained tough, dark brown bile. The spleen was dense, of a dark reddish-brown color, and tolerably large. The kidneys large, and very tough. Ramifications of veins, much filled with blood, were observed on the ileum.

Both lungs were, for the most part, attached by cellular adhesions to the costal wall; the cavities of the pleural sacs contained about a pound of serum; both lungs were puffy, and œdematous; in each of the lower lobes there was a spot of the size of a pomegranate, in addition to several smaller ones, of a blackish-red color and fragile,—a hæmorrhagic infarctus. The mucous membrane of the trachea and the bronchi was bluish-red, and loosened; and the bronchial ramifications were filled with a thick, yellowish, puriform mucus.

There were about two ounces of clear serum in the pericardium. The heart was more than twice the normal size, and invested with numerous milk-spots; the muscular substance was tough throughout, and of a reddish-brown color. The left ventricle was much dilated, and its wall was about an inch in thickness; and the right ventricle and the left auricle were dilated and hypertrophied. The valves were normal; the Foramen ovale was closed; the Venæ cavæ, the intercostal veins, the jugular veins, &c., were dilated and swelled.

The Aorta.—The preparation consisting of a part of the ascending aorta, the arch, and a portion of the descending aorta, presented the following appearances:

The ascending arch of the aorta (regarded as the vascular trunk designed to supply the head and the upper extremities) was unusually extended downwards; after giving off the arteria innominata, it diminished so much that its diameter did not exceed $5'''$ at the point where the left subclavian was given off, which formed, as it were, a continuance of it, and was of equal calibre with it. Above the valves its diameter was $11'''$. From this point it was deflected rapidly, and almost angularly, as a vessel of about $11'''$ in length, and not more than $3'''$ in diameter; its lower extremity corresponding to the depression of the obliterated Ductus arteriosus, was contracted and already undergoing obliteration, and was cut off from the descending aorta by a deep furrow.

At this spot, the calibre of the artery scarcely measured one line; the passage, which only admitted a thin probe, was obstructed in the direction of the descending aorta by a small plate of white, opaque deposit. The descending aorta varied from 8 to 9 lines in diameter.—The deposit was very considerable, opaque, and partially ossified, in the ascending aorta; the walls were rigid. The descending portion exhibited only a few plates of an opaque deposit. The arteria innominata was about 5 or 6 lines, the left carotid about 2 inches, and the left subclavian, as we have already remarked, about $5'''$ in diameter. The ends of the intercostal arteries branching off from the descending aorta, more especially the uppermost ones, were considerably dilated.

2. *Contraction and Obliteration in consequence of Disease of the Coats of the Vessel.*—To this class belong:

a. Obliteration of the mouths of a vessel, occasioned by the excessive formation of a tissue, analogous to the lining membrane, within a trunk.—This condition, which is followed by atrophy of the vessel itself, is a mode of obliteration of the arteries which has not been much regarded, and one whose nature is not known. It is the result of the excessive process of deposition in one of the trunks of the vessel, and in the vicinity of the mouth, as has already been described at p. 199. The mouth continues to become narrower, until it is finally closed by the last layer deposited around it, whilst it diminishes by the fusion of the mass around the circumference. After this the mouth very frequently appears as if it were closed by a membrane stretched across it. When the vessel has become shrivelled and wasted, the closed mouth presents a cicatrix-like puckered appearance, or has wholly disappeared. Above the closed mouth, the blood carried by the collateral circulation into the vessel coagulates over various extents of surface, until its coagulation is prevented by the circulation established by an anastomosing process. The artery shrivels and becomes atrophied above this clot or plug.

As this process of deposition must be very highly developed in order to produce such occlusions, and as it results in dilatation of the diseased artery, we are able to explain the appearance of these contractions and final obliterations of the mouths of the vessels, more especially upon the branches going off from aneurismal vessels. (See p. 215.) We have already alluded generally to the importance of this obliteration, which is, indeed, self-evident; but it exhibits special interest in some individual cases, among which we may notice the following:

1. The contractions and obliterations of the branches of vessels passing from the arch of the aorta.
2. The contractions and obliterations of the coronary arteries of the heart.

Neither of these is by any means a phenomenon of rare occurrence.

b. The contraction and final impermeability of an artery in consequence of excessive deposition—of its ossification—or of the deposition of fibrinous vegetations on the rough inner surface of the vessel, and their cretefaction.—This may be especially observed where the process of ossification is much diffused on the smaller branches of the femoral arteries; many cases of senile gangrene are based on this impermeability of the arteries, which, however, is seldom observed in vessels of considerable calibre.

3. *Occlusion of the Arteries. To this class belongs the occlusion of the vessel arising from different varieties of coagulation of blood.*

a. Occlusion of an inflamed artery.—According to our definition of arteritis, this condition can only affect arteries in which the circular fibrous coat is only subordinately developed. (See p. 193.)

b. Occlusion of an artery arising from a coagulation of the blood, depending upon an internal cause, such as a blood-disease.—To this class belong Velpeau's case of closure of the aorta, from the third lumbar vertebra downwards, with a part of the iliac artery, owing to a coagulation of a cancerous character in an individual exhibiting cancerous cachexia, and undoubtedly also the cases of occlusion of the thoracic and abdominal aorta observed by Schlesinger and Barth. Occlusion arising from arteritis, and especially the form above described, is very rare when compared with the frequency of occlusion of the veins.

4. *Contraction and obliteration arising from persistent pressure on the artery.*—Such a continued pressure may be exerted by different tumors, as goitres, encysted tumors, cancerous products, and aneurisms of neighboring arteries. Complete obliteration is very rarely induced by these causes; at any rate, in the larger arteries. The vessel becomes obliterated at the spot exposed to pressure in consequence of the coalescence of the lining membrane of the vessel; above this point the occlusion is affected by means of a plug reaching to the nearest branch, and beyond this the vessel is finally obliterated in the same manner as after tying the artery, as we shall have occasion to revert to in the sequel.

Besides these different modes of contraction, occlusion, and obliteration, we further noticed the following conditions when treating of aneurisms. (See p. 216.)

a. An impermeability of the mouths of the branches passing from an aneurismal vessel in consequence of their contraction into fissure-like openings.

b. An impermeability of the mouths of these vessels, induced under certain conditions already indicated at the above page, *by the fibrinous layers filling the aneurismal sac*, and the shrinking and obliteration of the vessel consequent upon it.

We shall consider the establishment of the circulation consequent on the obliteration of an artery when we treat of the healing of cut arteries, and the process of obliteration that follows the tying of an artery.

§ 5. *Mechanical Separations of Continuity.*—To these belong *lacerations and wounds of the arteries produced by cuts, thrusts, or gun-shot wounds.*

Spontaneous lacerations are the most important of any, especially those of the trunk of the aorta. To this class belong *lacerations of the large arteries arising from violent concussions or shocks*, viz., in consequence of a contusion (as, for instance, by a spent ball), striking a circumscribed portion of the vessel. No special interest attaches itself to those lacerations of the arteries which arise from excessive extension; such as, for instance, in the arteries of the extremities from dislocations, and which may be associated with extensive lacerations of the soft parts, and destruction of the bones.

Wounds of the arteries inflicted by cuts, thrusts, and shots, derive importance from the conditions of *false* and *varicose aneurism*, in which they frequently result.

A. *On the lacerations of the larger Arteries.—Dissecting Aneurism.*

Lacerations of the larger arteries, arising from *traumatic influences*, as from concussions and contusions of the body, are only interesting in a scientific point of view, when the different mechanical modes of laceration affect indifferently the separate arterial coats, and when they resemble certain spontaneous lacerations. Such is the case when the laceration implicates the two inner coats of the vessel (the lining and yellow membrane), while the cellular sheath of the vessel is in a state of integrity, or when the separation of its continuity does not correspond in extent, form, and direction with this laceration.

In this respect we must regard with special interest the *læsiones continui*, which are owing to some influence limited to a circumscribed portion of an artery, or to a loosening of the cellular sheath and a laceration of both its inner membranes owing to the same influence. They present the greatest similarity with that spontaneous laceration which is termed *dissecting aneurism.*

Spontaneous lacerations[1] may be classified under the following heads.

1. *The laceration depends upon a delicacy of construction of the whole arterial wall, and on the generally simultaneous narrowness (insufficiency of calibre) of the vessel; or on congestion, or excessive expansion of the mass of the blood.* We have observed several cases belonging to this class.

2. *The laceration depends upon a diseased condition of the texture of the coats of the arteries.*

The cases belonging to this class form two distinct series.

a. In those of the first series, the læsio continui consists in a detachment of the cellular sheath from the tube of the vessel, and of a laceration of the middle and lining coats of the vessel within the detached cellular sheath. The question here arises, which of the two is the primary and at the same time the controlling cause?

Experiments prove that it is by means of the cellular sheath, more especially of its elastic longitudinal stratum, that the artery is able to resist any violent lacerating action, and to sustain the force of the blood-wave when the texture of the inner layers, particularly of the yellow coat, is in a state of integrity.

In the above cases, the alteration of texture consists essentially in a chronic inflammation of the cellular sheath, which causes it to be more easily detached. The cellular sheath is here loosened, over various extents of surface, from the tube of the vessel, either alone or with an adhering layer of the yellow membrane, which is generally torn transversely, and only very seldom longitudinally to the vessel. The yellow coat is very brittle in the cases to which we refer, and where this condition was certainly the result of advanced age, this membrane admitted readily of being separated. The lining membrane was for the most part diseased, although only in a moderate degree, exhibiting a deposit which was partially ossified.—By way of elucidation we will give a case borrowed from the memoir before referred to.

A. G. v. P—, aged 52 years, a widow, fell to the ground in the street, on taking a quiet walk after dinner, towards evening, on the 18th of February, 1833. After being bled, she was carried to the hospital. She vomited twice, and after momentary recurrences of consciousness, died on the following morning, after long-continued and profound syncope.

Autopsy.—The body was of moderate size and thin.

The walls of the cranium were 3–4‴ in thickness and compact; on the left parietal bone, above the semi-circular line, a compact exostosis was discovered, about the size of half a walnut; the inner cerebral membranes were infiltrated. Some of the arteries on the base of the brain were partially ossified.

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[1] Oesterr. med. Jahrb., Bd. xvi. St. 1.

The lungs, with the exception of the swollen anterior margins of their upper lobes, were of a dark red color, rich in blood, and œdematous at different points. The left pleural cavity contained 1 lb., and the right cavity about 4 oz. of pale reddish sanguineo-serous fluid.

There was considerable extravasation of coagulated blood in the posterior mediastinum round the aorta and the œsophagus, more especially, however, round the pulmonary vessels and the branches of the trachea, towards the roots of the lungs; the pericardium contained upwards of a pound of coagulated and fluid blood.

The heart was somewhat larger than usual, nearly of a round form; the left ventricle and the Conus arteriosus were very large, and the walls of all the cavities were of normal thickness. The right side of the heart was covered with a considerable layer of fat; the ramifications of both the coronary arteries were, for the most part, ossified. The substance of the heart was pale and friable.

Aorta.—The ascending aorta, like the pulmonary artery, was very wide; the valves of the former were thickened at their insertion and their nodules, and were partially ossified. The cellular sheath (the elastic and cellular coats) of the aorta was loosened throughout its entire length round the ascending portion, at its arch and on the whole of the thoracic and abdominal aorta, over full a third of its circumference, where there adhered to it either a thin layer or partially exfoliated thicker portions of the yellow coat. This condition extended upwards over the arteria innominata to the common carotid, the right subclavian and its larger branches, and downwards over a portion of both the iliac arteries; in the former the cellular sheath was entirely separated over the whole circumference of the artery, while in the latter it was only partially loosened, or admitted of being easily detached all round, together with the external layer of the middle coat. The same condition was observed in most of the small and large branches of the thoracic and abdominal aorta for a considerable extent of surface from their points of departure. The cellular sheath was of a bluish-red color, infiltrated with blood at many parts of its detachment, and very thick; at those points, however, at which a layer of the yellow coat still adhered to it, and where the two were not entirely separated, it was paler and less thick from a deficiency in the suffused blood, and was intersected by a highly developed network of vessels. The free space between this and the yellow coat of the vessel was filled with a considerable quantity of coagulated and fluid blood.

Within the cellular sheath, which was detached, as we have already seen, from the ascending aorta, the yellow and lining membranes were torn transversely over an extent of an inch and three-quarters above the valves, so that there remained only a spiral strip of their posterior wall (about two lines and a half in breadth, and equal in length to about half the circumference of the aorta), which connected together the two extremities of the rent, and was raised along the concavity of the trunk of the aorta from its original horizontal position, in consequence of the displacement of the upper extremity of the rent, which we shall now proceed to notice.

While the lower extremity of the rent was turned upwards with an almost circular opening, in consequence of the exposed transversal rent, the upper one was almost entirely enclosed by the convex wall and driven into the cavity of the arch of the aorta as far as the left subclavian, the entrance of which was even obstructed by a conically rolled portion of the tube of the aorta, so that a communication was opened from the subclavian into the inserted vessel, and through this into the cavity of the cellular sheath.

In this manner, both the extremities of the rent were from about an inch and a half to an inch and three-quarters from each other, while within the almost saccularly expanded cellular sheath at this spot, as well as over the whole extent of the aorta and the branches already referred to, the space between it and the yellow coat of the artery was completely filled with coagulated and fluid blood. This accumulation of blood had compressed the aorta and the cœliac axis at different points, and completely detached from their origin several small branches of the aorta and a large branch of the renal artery on the left side.

The blood had been further extravasated from this space into the pericardium and the mediastinum, in the following manner. The cellular sheath of the ascending portion of the aorta was torn outwards and backwards along the descending Vena cava, near its opening into the auricle, in a longitudinal direction, together with the portion of the pericardium by which it was invested. This sheath was also considerably attenuated at several points along the descending aorta, where it readily admitted of being torn.

The yellow coat of the artery could be easily peeled off in all parts, but more particularly at the aorta itself; it was also very brittle. Several small bony plates were observed in the lining membrane of the arch of the aorta.

The intestines were pale throughout, although this pallor was especially perceptible at some circumscribed portions of the ileum where the mucous membrane was perceptibly attenuated, and had even wholly disappeared.

When we consider the appearances here presented, with a view of ascertaining the relation of the different coats of the artery in their physiological and pathological condition, we arrive at the following theory, viz., *that a detachment of the cellular sheath occurs spontaneously at a certain stage of its disease, giving rise at the same time to laceration of the two inner coats.* These coats are usually torn transversely along the course of the yellow fibres, in consequence of the artery being deprived, at the moment when the cellular sheath is detached, of the support which had limited its further expansion and stretching. Such a laceration is also the more readily effected, when the two inner coats, notwithstanding the integrity of their texture, are unable to resist this expansion and tension from having become soft and brittle, owing, as is commonly the case, to advanced age, or to the dilatation of the vessel which is observed in all such cases.

The detachment of the cellular sheath must, therefore, constitute the primary agent or cause, while the laceration of the inner coats is the consecutive effect of this condition.

It is, however, probable that this may admit of a different explanation. Thus, for instance, we are unable to apply this theory when, in addition to the cellular sheath, an adhering layer of the yellow coat is loosened with it at the spot of the laceration, and when, therefore, the rent itself affects only the inner layer of this coat (with the lining membrane of the vessel). Another theory suggests itself when we consider that dilatation of the vessel is present in all cases. *This dilatation depends, in all probability, upon the paralysis of the elastic layer, owing to a chronic inflammation of the cellular sheath; and the laceration of the yellow coat of the artery might therefore be the final result of the greatest dilatation it was capable of resisting without any considerable disturbance of texture of the whole arterial wall.* Laceration will, moreover, be the more readily effected in proportion to the brittleness of the yellow coat dependent on the advanced age of the patient.

In accordance with this view, the rent in the lining and yellow coats must be the primary occurrence, and the loosening of the cellular sheath, either with or without an adhering layer of the yellow coat, must be regarded as a secondary result owing to the forcible escape of blood from the rent. The following conditions appear from our observations to be worthy of notice as controlling causes:

The heart is hypertrophied in all cases, and its left ventricle is in a state of active dilatation. In most cases the laceration is effected without any special excitement of the heart's action, so that the occurrence must be regarded as the final result of the diseased condition of the vessel.

The integrity of the detached cellular sheath, that is to say, the hinderance thus opposed to the free extravasation of the blood, occasionally postpones the fatal termination for a few hours; in Laennec's case death was delayed for four days.

The cellular sheath is generally lacerated in consequence of its distension by extravasated blood, usually in the vicinity of the lining membrane of the artery, but occasionally, however, at one or more spots remote from that rent.

These lacerations are generally *transverse*, and only rarely take a longitudinal direction.

Lacerations are much more frequent in the ascending aorta, at a short distance above the valves, than in the thoracic aorta.

They generally occur in persons of advanced age.

This form of lacerations belongs to the class which has been repeatedly investigated by English pathologists, who have applied to them the inappropriate designations of *dissecting aneurisms*, or of *anomalous* or *interstitial aneurisms*. These observers have not hitherto given a feasible explanation of this process, and they appear to have overlooked the conditions that induce such diseases of texture.

b. The cases belonging to the second series differ in every respect from those of the first. *These are lacerations of an artery exhibiting a profoundly diseased condition of the texture of the whole wall*—somewhat in the manner of the so-called *dissecting aneurism*, that is to say, *with* *detachment of the cellular sheath, but this is here always effected by the violent action of the blood extravasated from the rent, and therefore constitutes a secondary occurrence.*

A cause predisposing to these lacerations is afforded by a high degree of the disease which we have described at p. 199, and to which we have referred as the cause of origin of aneurism. The cellular sheath of the vessel here firmly coalesces with the yellow coat of the artery, in consequence of a process of chronic inflammation by which its tissue becomes thickened, callous, and condensed.

The inner coats of the dilated artery are lacerated in consequence of their morbid brittleness within the closely adhering, thickened, callous, resistant, cellular sheath, which is here violently detached by the blood, but never over an extended surface, as in the cases belonging to the first series.

The cases belonging to this class are generally *longitudinal lacerations*, in which the fibres of the yellow coat of the artery are actually torn asunder. Transverse lacerations occur only as exceptions to the rule. When laceration takes place after a very considerable degeneration, with unequal disease of the arterial coats, the rents are irregular and curved. The following case may serve as an illustration of these appearances.

On the 6th of March, 1834, a post-mortem examination was made of the body of a woman, aged 50 years, who had died suddenly two nights before. The autopsy showed the body to be robust, and in tolerably good condition. Both arms bore marks of repeated venesection.

There was a grayish white foam, collected in the trachea.

The lungs were of a dark-red color, very full of blood, and œdematous, excepting in the right lower lobe.

There were two pounds of coagulated and fluid blood in the pericardium. The heart was half as large again as usual, fat, and flabby in the left ventricle, and the Conus arteriosus of the right ventricle was dilated. The auricles and trunks of the vessels contained coagulated and fluid blood.

The ascending aorta and its arch were considerably dilated; their inner surface was uneven, and covered at some parts with a white, opaque, cartilaginous, and smooth deposit, and at other parts with a light-colored, wrinkled deposit of considerable thickness; the mouths of the three branches of the arch of the aorta were contracted. About an inch and a half above the semilunar valves on the concave wall of the ascending aorta there was a jagged, rectangular rent in the diseased inner and middle (yellow) coats of the artery. The longest direction of the rent measured one inch and five lines, and ascended into the arch of the aorta, while its other side (which was only half the length of the former) extended along the posterior wall of the aorta. A rectangular lobule, composed of a portion of the inner and of half the thickness of the middle coat, had been exfoliated from the above-described right angle, and from this point the ascending aorta had lost the cellular sheath, together with the external layer of the middle coat, except at a mere narrow strip on its concave surface. The space between these two laminæ was filled with coagulated blood. The external lamina had burst into the pericardial cavity backwards, behind the descending Vena cava, longitudinally and downwards, over a surface extending more than half an inch, together with the contiguous lamina of the pericardium.

The cellular sheath of the aorta was unusually thick, although at the same time of a callous condensation, and intimately connected with the yellow coat. It was of unusual thickness, nearly $1'''$ at the arteria innominata, and more especially at the right subclavian, and was converted into a whitish, very dense and tough, lardaceo-fibrous stratum, and fused as it were into the yellow coat. It was less thick at the left carotid and the subclavian, although it presented a similar character.

On examining the abdominal cavity, the gall-bladder was found to contain a concretion, about the size of a nutmeg; and the fundus uteri was filled with a fibroid growth, equal in size to a child's head, and attached by a thick pedicle.

These Lacerations, like the diseases in which they originate, generally occur in advanced periods of life. They also usually affect the ascending aorta, which may be explained by the circumstance, that this vessel is, in most cases, especially diseased, while it is at the same time exposed to the force of the blood-wave propelled from the heart.

The heart, as may be conjectured from the observations already made, is subject, in these cases, to dilatation and hypertrophy, more especially of the left ventricle.

Among the aneurismal forms, especially allied to these cases of the second class, we must reckon *lacerations of the smaller, diseased arteries, having rigid membranes and having become brittle, which are either frequently spontaneous, or the result of wholly inexplicable conditions, such for instance as we especially see in apoplexy* (cerebral hemorrhage).

3. *Finally, this laceration may depend upon the removal of the supports of an artery, in consequence of an ulcerous process, and upon a loosening and softening of its texture, arising from its coats becoming infiltrated with the ulcerous secretion surrounding them.* This form of laceration more particularly affects delicately constructed arteries of inferior calibre, as, for instance, the branches of the pulmonary artery in the walls of tuberculous pulmonary caverns. In some few cases the laceration is preceded by a lateral (aneurismal) enlargement of the vessel towards the cavern (see p. 198).

B. *On Incised, Penetrating, and Gunshot Wounds of the Arteries.*

Such injuries of the artery as are inflicted by sharp-pointed instruments, even where it is only opened at the side, and shot-wounds which merely remove a small portion of the wall of an artery, are, as is well-known, extremely dangerous; for they usually give rise to the so-called *false aneurism*, and, under certain conditions, to *varicose aneurism*, which we shall soon consider in detail. It is true that penetrating and incised wounds of an artery may heal under favorable conditions, as we see in cases where the temporal artery has been opened, and, as Amussat has recently shown, by observations at the bedside and by experiments on animals, in the same manner as similarly injured veins. But as in man, injuries are often inflicted on the arteries under circumstances which exclude the concurrence of these favorable conditions, such wounds do not commonly heal; in gunshot-wounds of the artery more especially, a cure is never effected by the adhesion of the margin of the wound, but, as an ordinary consequence, we generally have the so-called *false aneurism.*

On False Aneurism.—When an artery of one of the extremities has been injured in any of the above ways, the blood is effused into the surrounding cellular tissue, forming an extravasation, if unable to escape from the outer wound. The blood is then accumulated in a cavity formed by the laceration of the tissue, the structures around it being suffused and infiltrated with blood. This constitutes *diffuse false aneurism*, or, according to Foubert, *primary false aneurism.* When considerably diffused it in general terminates fatally in inflammation degenerating into gangrenous disintegration, associated with symptoms of paralysis, and in continuous external hemorrhage. It is only in rarer cases, and when the aneurism is less diffused, that it can heal by the artery becoming obliterated during the ichorous process, through arteritis, and by the drying up of the ulcerous process, after expulsion of the extravasation and of the tissue destroyed by it.

When the neighborhood of the extravasation becomes the seat of an inflammatory process (reaction), tending to condensation (sclerosis) and hypertrophy of the tissue, the cavity containing the extravasation acquires a true wall and definite limits, and becomes converted into a sac, seated upon and surrounding the artery, and into which the arterial wound opens. A lining membrane may be formed upon its inner surface, and the sac may then present such similarity with a mixed *aneurism* as to render its anatomical diagnosis extremely difficult. In this condition, the collective appearances represent what is commonly known as *false circumscribed*, or Foubert's *consecutive false aneurism.*

It is obvious that these conditions do not originally merit the designation of aneurism, whilst the consecutive condition of a *false circumscribed aneurism* presents appearances which give it in every respect the significance of an aneurism. This form of aneurism, which commonly attains a very large size, as, for instance, in the popliteal space generally, as is the case with large aneurisms, terminates fatally when left to run its course.

On varicose aneurism, Aneurysma spurium varicosum, Varix aneurysmaticus, A. per anastomosin (W. Hunter), *A. per transfusionem* (Dupuytren).

This aneurism consists in the communication of an artery with a neighboring vein, effected by means of an aperture in the artery corresponding to one in the vein. This communication may be *direct* or *indirect*, and further may be the result of injury, or may occur *spontaneously.*

Varicose aneurism is most commonly produced by some injury which simultaneously affects the contiguous walls of an artery and of a vein; such, especially, as penetrating wounds or injuries from small shot, and when it arises from incised wounds, it is in general owing to venesection in which both walls of the vein have been cut through, and the lancet has penetrated through the wall of the artery below it. The latter mode of injury is, moreover the most frequent cause of *varicose aneu-*

rism, and consequently the bend of the elbow is the most common seat of this aneurismal formation. Moreover, neighboring arteries and veins may be so much injured by splinters of bone that the arterial blood may enter a vein. The same result has also been effected by violent contusions.

The most common form of traumatic varicose aneurism is that occurring after venesection, and seated between the brachial artery and the median vein, or when the brachial artery divides higher up, between the radial or ulnar artery and the median, cephalic, or basilic vein. The same form of aneurism has also been observed in the brachial artery in the upper arm, in the subclavian, in the carotid with the jugular vein, in the femoral, popliteal, temporal, and other arteries.

Spontaneous varicose aneurism is the opening or rupture of an aneurism into a vein that has coalesced with it. Cases of this kind have been noticed by myself and many foreign observers in the femoral artery, in the abdominal aorta with the Vena cava inferior, and in the ascending aorta with the Vena cava superior.

The communication established between the artery and the vein is, as we have already remarked, either *direct* or *indirect*. The former is frequently observed at the elbow, as a consequence of venesection; thus, for instance, whilst the outer wound of the vein is cicatrizing under a bandage and compress, which prevent the formation of an extravasation into the cellular tissue, the two vessels coalesce together, more especially in the immediate vicinity of the openings of the wounds, by which means a direct communication is formed between them. The vein at the spot presents the appearance of a swelling or roundish expansion, which commonly increases to about the size of a hazel-nut or half a walnut, but, in some rare cases, attains an extraordinary volume (Hodgson, Larrey). The direct communication of the two vessels, and the dilatation of the vein at the corresponding point constitute what is known as *aneurysmal varix*.

Such a communication between the artery and the vein is always present in spontaneous varicose aneurism.

The *indirect mode of communication* is effected in the following manner by the presence of a false aneurism. In those cases in which a sufficiently strong compress has not been applied immediately after the injury, and where two vessels have been injured, which are not in immediate contact, or are not fixed in one common layer of cellular substance, or where finally the wounds in each do not originally correspond together, or where, after the injury has been inflicted, the vessels have been displaced or separated,—an extravasation into the cellular tissue is formed, which, in the last-named cases, pushes the vein aside from the artery, and thus prevents the establishment of a direct communication between the two.

This extravasation,—a false diffused aneurism,—is now reduced to a false circumscribed aneurism, the cavity of which forms the medium of communication between the artery and the vein. This false aneurism varies in size, but it commonly is not larger than a walnut or a hen's egg. It generally forms a more considerable and a tougher swelling than that which occurs in a direct communication, and hence Scarpa was led to distinguish this condition from aneurismal varix by the designation of *varicose aneurism.*

The aneurismal sac presents many differences in reference to the extent to which it lies on the artery, and especially in relation to the opening.

The size of the openings into the two vessels also varies very considerably, and depends, like their form, on the size of the original wound, on the instruments by which the injury was inflicted, &c. The opening in the artery in general permanently retains its original size and form, whilst that in the vein undoubtedly experiences many alterations like the aneurismal sac itself.

The openings in the vessels, as well as the inner surface of the aneurismal sac, are invested with a lining membrane of recent formation, which continuing into the two vessels, gradually loses itself, and merges, more especially in the case of the vein, into the deposit formed upon the inner surface of that vessel. In consequence of this, the openings in the vessels have a smooth and healed appearance.

In the so-called aneurismal varix and in spontaneous varicose aneurism, the communication is effected by means of a simple opening, through the coalescing adjacent walls of both vessels, which acquires a smooth and healed appearance in consequence of being invested with a membrane of recent formation, and thus loses its original character of a rent or rupture.

All observers are unanimously of opinion that the arterial blood principally, if not exclusively, passes into the vein, in both these modes of communication, the direct as well as the indirect. Breschet thinks, that in an operation for a varicose aneurism he also saw the venous blood pass into the artery through the aneurismal sac, during the diastole of the latter. To this circumstance, which may indeed occur in some cases, he refers the dilatation of the arteries below the aneurism, together with the attenuation of the arterial walls, in consequence of their contact with venous blood,—an explanation that has been given by no other observer.

The character of the vessels above and below the place of communication, as indicated by all observers, and as I have repeatedly had occasion to notice, corroborates the existence of a very preponderating current of arterial blood towards the vein. The vein becomes first dilated below the point of communication, and then finally spreads beyond and above it. This dilatation is associated with an enlargement of the valves. The walls of the veins become thicker and more rigid, principally in consequence of the formation of new layers of lining membrane (see p. 205); they at length acquire an appearance similar to that of the arteries. Below the communication the artery is contracted in consequence of the blood being drawn away towards the vein; and its walls then become thinner, relaxed, and more similar to the veins in consequence of the diminished energy of their function, corresponding to the extent to which the blood is drawn away from it. The artery becomes dilated above the communication, in consequence of the obstacle which the venous blood opposes to the entrance of arterial blood into the vein.

The limb below a varicose aneurism is frequently swollen; it also presents a cyanotic color, its cellular tissue is infiltrated and hypertrophied, and the general investment is the seat of repeated erysipelatous inflammations, excessive epidermal formation, &c.

The sequelæ of spontaneous varicose aneurism between the trunks of the aorta and of the Vena cava are obvious, but they are often indistinctly manifested in consequence of their being masked by the results of the aneurismal affection of the trunk of the artery, and by the simultaneous presence of heart-disease.

The process of healing and obliteration after arteries have been cut through or tied.—An artery, on being cut through, is immediately retracted in its sheath,—at any rate, as far as the next lateral branch, if of considerable size, while it at the same time becomes gradually contracted. The blood pours outwards in a greater or less quantity, according to the extent and position of the external wound, or it is effused into the cellular tissue. Independently of the aid afforded by art, the exhaustion itself exerts a favorable action on the coagulation of the blood effused around the artery. The coagulum within the canal of the arterial sheath, which is produced by the retraction of the vessel, forms the *outer plug,*—the most essential and the actual means of arresting the hemorrhage. In addition to this plug, another—the *inner plug,* is gradually formed within the artery itself, by the coagulation of the blood which is arrested in the stump by the first plug, unless a considerable lateral branch is given off in the immediate neighborhood.

By these means the necessary conditions are obtained for arresting the hemorrhage; and the cure—closure—is then effected by the adhesive inflammation of the cellular sheath of the vessel on the margin of the wound and by the obliteration and final atrophy of the whole stump of the artery, as far as the next collateral branch, in the same manner as we observe after ligature, torsion, &c., and which we will now proceed to consider.

The whole healing process of arteries that have been cut through, has been fully elucidated by the invention of the ligature, and by the numerous investigations in reference to this process, as shown by experiments on animals. The labors of Stilling have thrown the greatest light on this subject in modern times; and we purpose in the following remarks to borrow from them the most important facts, which we will incorporate with our own views regarding individual points of the whole process, as obtained from investigations on the human subject. We would here briefly remark that the alterations resulting from ligature, &c., more especially the formation of thrombus, are more evident at the cardiac than at the peripheral end of the affected artery, and that the following remarks more especially apply to the alterations effected at the former of these points.

After the application of the ligature or torsion, the terminations of the cut artery, as has been already observed, retract. This gradual contraction, which affects the artery as far as the next lateral branch, probably depends, at first, upon the irritation set up by the ligature, and subsequently, on the decrease in the quantity of blood entering this portion of the artery, in consequence of its abstraction towards the dilated branches, and on the diminished impulse. By this contraction of the fibres of the yellow coat the lining membrane of the vessel is wrinkled into delicate longitudinal folds; whilst the whole of the wall of the vessel is puckered into more considerable plaits at the spot where the ligature has been placed.

The ligature or torsion takes the place of the external plug, causing a stoppage of the blood in the vessel between it and the first lateral branch. By being arrested, it forms a conical coagulum, that is to say, in the words of Stilling, "the arrested blood forms a conical mass, whose apex is near the first lateral branch, and whose base is seated on the extremity of the vessel, and is contained within a funnel of blood in partial motion. The larger opening of the funnel, where its walls are sharply cut and very thin, is in the neighborhood of the base of this cone, or just above the extremity of the vessel, whilst its smaller (blind) opening, whose walls are constantly increasing in thickness, until they finally unite, lies near the first lateral branch, in the axis of the vessel or the middle of its cavity." This conical coagulum constitutes an inner plug, which is commonly designated a *Thrombus.*

The thrombus begins to be formed at the termination of the vessel, and from thence extends onwards in its axis to the point where the first lateral branch is given off; it possesses a conical shape from its commencement, and not being very thick, it does not entirely fill up the tube of the stump of the vessel, but simply projects into it with its base or middle, at which parts it is usually of a dark, blackish-red color, and of very inconsiderable consistence. The apex, however, is white, more dense and hard, and resembles coagulated fibrin. It occasionally acquires a more fusiform shape by the addition of supplementary new layers, consisting in such cases of concentric superficial strata in addition to the original coagulum in the centre. It occasionally lies free in the stump of the artery, but more commonly it adheres, although at first loosely, by its base. It is subsequently invested with an albuminous moisture, by means of which it adheres loosely to the wall of the artery, although its apex remains free. This adhesion is frequently effected by means of filamentous bridge-like attachments.

The inflammation, which is set up around the injured vessel, also implicates its cellular sheath. Plastic lymph exudes into the tissue of all the structures and into the cellular sheath of the vessel. We find also that a process of adhesive inflammation affects the spot at which the ligature is applied, and where the different folds of the lining membrane of the vessel come in contact with each other; and that there is an exudation of coagulable lymph, which causes a slight adhesion of the walls of the vessel, and of the thrombus at its base. The inflammation in the terminal part of the vessel is owing, not only to the irritation established by the operation, but also to the thrombus, which acts here as a foreign body; exudation being effused between the coats of the vessel as well as also on the free surface of the lining membrane.

The thrombus is always formed gradually; but in some cases it is found to be forming within half an hour or an hour after the operation, while, in other cases, there is no trace of it at the end of several hours. It is generally completely developed within twelve or eighteen hours after the closure of the vessel; it is more rapidly formed in small than in large vessels.

The further alterations include the metamorphosis of the developed thrombus, its coalescence with the wall of the vessel, and the final obliteration and atrophy of the artery.

The thrombus occasionally exhibits light-colored spots, both on its surface and in its interior towards its apex. Stilling observed fibrous or thread-like stripes on these spots, which he convinced himself by a lens were vessels.

In the course of time the thrombus acquires a porous structure, and becomes spongy and cavernous. Stilling found, in several experiments, that in addition to the numerous canals which he injected, and which traversed the thrombus in different directions, there was a central longitudinal canal opening into the cavity of the vessel. The periphery was especially injected in the more recent thrombi, whilst in those of older formation, the injection advanced more towards the centre or the axis. Where this so-called *vascularization* was present, the thrombus was always of a paler, flesh-like, faint rose-red color, turning to yellow, and finally to white, whilst the consistence was proportionally more considerable.—This metamorphosis of the thrombus is succeeded by its *regressive formation.*

In the meanwhile the thrombus becomes intimately adherent by its base, and very frequently by the whole of its body to the wall of the vessel,—in smaller vessels on the second or third day, and in larger ones on the fifth or sixth day; the former adhesion has now been converted into a firm coalescence. The greater part of the apex of the thrombus commonly, however, remains free during the period of its greatest vascularity.

The regressive formation of the thrombus consists in the diminution of the number of the so-called vessels within it, in its increased pallor and density, and in the fact, that "the whole mass of the thrombus still remaining at this period, merges, as it were, into the mass of the stump of the artery, forming with it one body." (Stilling.)

This portion of the vessel gradually loses its proper texture; the exudation effused into the coats of the vessel becomes in part resorbed, and is in part metamorphosed into a cellular or fibroid tissue; the walls of the vessel gradually close around the shrivelling thrombus, and become obliterated into a cellulo-fibrous, ligamentous string, which in the course of time disappears still more, until it can no longer be recognized. This alteration is effected in smaller vessels in from about twenty to twenty-two days, and in larger ones in from thirty to forty-five days.

The ligatures by which the middle circular fibrous coat and the lining membrane have been originally divided, are loosened, and come away, in consequence of the suppuration of the cellular sheath at the spot where they have been applied, during the above-mentioned process.—This suppurative process not unfrequently gives rise to destruction of the coats of the vessel above the ligature, and of the thrombus, and hence induces hemorrhage.

The circulation is established, after the application of the ligature, in the same manner as in obliteration of an artery generally, by the dilatation of the lateral branches and their anastomoses,—the so-called collateral circulation, which is developed, in cases of spontaneous and gradual obliterations, with a rapidity proportional to the increasing contraction of the vessel, so that its final occlusion is imperceptibly effected.

When a main artery has been tied, the circulation is at first carried on by means of all the innumerable communications of the small ramifications; subsequently, however, some of these vessels and anastomoses dilate in a preponderating manner, while the others gradually return to their normal calibre. This dilatation is especially remarkable in the small branches, whilst the trunks and larger branches are relatively dilated to a very inconsiderable degree (Hodgson.)

We would only add to this description the materials derived from a review of the facts and opinions which have been deduced from an investigation of tied arteries in man, and from the simultaneous consideration of highly important conditions analogous to thrombus.

We must, however, at once premise, that we do not regard this question as wholly settled, since this process in man presents numerous anomalies, independently of those cases in which the thrombus is not duly formed, in consequence of debility, cachexia, &c.

Our views in reference to the process, and the individual conditions on which it depends, are as follows:

1. We are of opinion, *that the occlusion of a tied vessel may take place without the occurrence of thrombus* (*the inner plug*), and that this is a mere incidental formation, and not by any means an inevitable and necessary condition of obliteration. There is very frequently no thrombus present, and occasionally its place is supplied by an adhering red gelatinous, in general irregularly thick, wrinkled, gland-like, shaggy coagulum, whose color is subsequently changed to a yellowish-red tinge; or the thrombus is inadequate to fill up the whole of the vessel, and hangs loosely in the stump of the artery, without actually adhering at any one point; although, notwithstanding this arrest of growth in the thrombus, it yet closes the extremity and a neighboring portion of the vessel. No trace of the previous existence of a thrombus can, however, be detected on cutting through this coalescence.

2. Although we would not wholly deny the point in reference to every case, we are yet of opinion *that a true arteritis, with exudation on the inner surface of the vessel—constituting the so-called adhesive exudation, by which the thrombus is fixed and made to adhere to the wall of the vessel—is not an essentially necessary condition;* since we have found it absent in numerous cases, in which all the requirements for occlusion were present,—that is to say where neither a change, induced by exudation in the tissue of the circular fibrous coat and in the lining membrane of the vessel, nor a free exudation on the latter could be distinctly recognized. We do not regard the albuminous or gelatinous layer, which invests the thrombus and the lining membrane of the vessel, and attaches the thrombus to the arterial wall, and which at first is transparent, but subsequently becomes white and opaque, as the product of arteritis, or as an exudation, but as a product of the blood—as a structure analogous to the lining membrane (see p. 204), which is produced in the stump of the vessel with a readiness proportional to the necessary mechanical conditions which are present. We may very often distinctly perceive

how it encloses the thrombus, and extends, sometimes in a bridge-like form, from its basis towards the wall of the vessel. In other more advanced cases, these two lamellæ are everywhere, or at some spots fused together; and, in the latter case, as the thrombus does not completely fill the vessel, it adheres by threadlike structures or bridges. The wrinkled, velvety coagulum investing the inner wall of the vessel, and which we have already described, has a similar significance; in like manner we believe, that the degeneration which attacks the circular fibrous coat, and gives rise to loosening, bleaching, and lacerability, is not to be regarded as the consequence of an exudation, and to be referred to its action on the tissue, but must be considered in the light of a regressive metamorphosis—an involution,—such as we meet with in atrophies of arteries, as, for instance, the obliteration of the fœtal passages, &c., which are effected without the agency of any inflammatory process.

3. We hold that *the occlusion and obliteration of tied arteries are essentially dependent on the same process that occurs in vessels which no longer receive an energetic current of blood, in consequence of the circulating fluid taking another course, and become unserviceable*, as for instance, the umbilical arteries and the Ductus arteriosus. After the end of the tied artery next the ligature has become closed by the fusion of the opposite surfaces of the inner wall of the vessel, the further obliteration follows from the decrease of the vessel as the blood is turned into another course, and a collateral circulation established; and from its walls finally coalescing, either by means of the original lining membrane, or of a newly-deposited layer of that structure. The white mass, which we find as a central substance in the stump of the vessel, seems therefore to consist of this newly-deposited stratum.

When a thrombus is formed, which is far more commonly the case, the same process takes place—that is to say, the vessel contracts, and becomes occluded above it; it undergoes a metamorphosis into a fibroid string—a white fibrous mass.

4. *Whether the thrombus, in certain cases, disappears by resorption into the mass of the blood in a state of minute disintegration or* (as Remak expresses it) *by solution, is a point which is certainly not at present established.* There are, however, no facts positively opposed to this view, and it would be an occurrence in whose favor there are many analogies,—as, for instance, the fusion or resolution of coagula of blood in inflamed veins, the diminution and the final disappearance of vegetations on the valves of the heart, the disappearance of the ends of phlebolites, &c.

5. We have never observed the formation of vessels in a thrombus (its so-called vascularization). We do not, however, in the least doubt the accuracy of Stilling's observations,—that is to say, that the mass of the thrombus was porous, and capable of being injected; we cannot, however, participate in his view, that this condition depends on a true formation of vessels, and represents an organization of the thrombus. We prefer believing that *this condition is the same as that with which we have become acquainted as channelling of the deposit* (*of the structure analogous to the lining membrane of the vessel, and formed in great excess*, see p. 200), and which we regard as a very remarkable pheno-

mena; that even this channelling sometimes occurs in other structures similar to thrombus, as, for instance, in the fibrinous coagula in the heart; and that it is of this, and nothing else, which observers speak, when they fancy they have injected polypi of the heart (Alex. Thomson, Vernois, see p. 166). We have recently had opportunities of observing this porosity, and the cavernous structure to which it gives rise, in vegetations within the cavities of the heart.

Holding this view of the case, we cannot regard the diminution and shrivelling of the thrombus, whereby its vessels—that is to say these canals—becoming obliterated, as a regressive formation in Stilling's sense.

6. Neither have we had an opportunity of observing *a long central bloodvessel, either single or ramifying at its extremity, running through the stump of the obliterated vessel*, as described by Lobstein and Blandin; nor have we ever observed the arborescent sprouting of vessels from a stump, as seen by Jones, Ebel, and others. According to our view, this phenomenon is intimately connected with the channelling of the thrombus, and the presence of these central vessels in the stump is exclusively owing to the persistence of longitudinal canals in the thrombus, such as have been frequently noticed by Stilling; and these arborescent vessels are nothing more than such persistent canals of the thrombus, which may perhaps, in the course of time, become longer and broader within the atrophying stump of the vessel. They most assuredly have no affinity with true vessels, however generally they may be regarded in that light.

We think it highly probable that Mayer's case of two arch-like lateral vessels, which connected the two extremities of the carotids after they had been tied, belongs to this class, although we are unable to give a definite opinion on the subject.

The formation of central canals in the coagula which obstruct inflamed veins—under which head we must include the case observed by Barth, of a central canal through an old plug obstructing the abdominal aorta, —may depend upon the same process of channelling, or upon another process, to which we shall refer, when we proceed to the consideration of the veins.

IV.—ABNORMAL CONDITIONS OF THE VEINS.

§ 1. *Deficiency and Excess of Formation.*

We have already noticed, under the head of Anomalies of the Heart, the most important anomalies and other deficiencies of structure affecting the trunks of the venous system. Moreover our remarks, in the corresponding chapter on the Arteries, apply likewise to the Veins.

§ 2. *Anomalies in their Origin and Course.*

Various anomalies of this nature are of frequent occurrence in the venous system, although they do not, according to Meckel, preponderate over those of the arteries to so extensive a degree as is generally supposed. We refer our readers to the more circumstantial anatomical works for a detailed notice of these anomalies.

There is, however, one form of anomaly belonging to this class which deserves especial mention, notwithstanding the notice which will be given of it under Dilatations of the Veins. This form consists in an anastomosis of the epigastric cutaneous veins with the umbilical vein at the navel, on which depend the persistence and patency of the latter vessel.

§ 3. *Diseases of Texture.*

We purpose, for the better comprehension of the subject, prefacing our consideration of other anomalies, as, for instance, those of calibre, by a notice of these diseases.

a. Inflammation.—The study of inflammation of the veins (*Phlebitis*) constitutes one of the most important departments of pathology. It is entirely the result of anatomical research; yet, however complete may appear to be the development which this subject has attained in our day, it still presents many important deficiencies, which have either been disregarded by observers designedly or from a deficiency of materials, or have been supplied by irrational conclusions and hypotheses.

Inflammation of the veins is a very frequent disease, and is highly important, both on its own account, and also more especially from the absorption of its products into the blood. It is, under all circumstances, incomparably more frequent than inflammation of the arteries.

Its seat is the cellular coat of the vein, and likewise the cellular fibrous coat, in as far as the latter exhibits a certain degree of vascularity; and its products are deposited alike in the tissue of both these coats, and in the non-vascular strata of the lining membrane of the vessel from whence they extend to the canal of the vein.

It more frequently exhibits an *acute* than a chronic character, and it is then distinguished by the deposition of exudation on the inner surface of the vessel. The following remarks refer to this form of the disease, the *chronic* form of which will be considered in a future page.

It is especially necessary to distinguish *two forms* of phlebitis:

1. *Phlebitis* (*inflammation of the coats of the veins*) *is the primary disease*, although it may be owing to various causes, *while every anomaly of the blood within the inflamed tube of the vessel, and still more, perhaps, beyond that spot, such, for instance, as the coagulation of the blood within the inflamed vessel, is a secondary phenomenon, depending upon the product of the inflammation.*

This phlebitis is very frequent as a *primary* disease, and arises from the most various injuries, as cuts or thrusts, affecting either the vein alone, or, conjointly with it, other soft and firm parts; from contusions and displacements of different soft parts including the vein; or from many forms of surgical or medical maltreatment of wounds of the veins. This disease so far depends upon the epidemic constitution, that it is of extremely frequent occurrence at certain periods, with or without the concurrence of these favoring circumstances. This disease may also be of a *secondary* character, and in that case it is derived either from inflamed contiguous structures—as, for instance, the inflammation of the veins in the neighborhood of abscesses, phlebitis from inflamed carious bones, &c.,—or is of a metastatic nature, as the phlebitis which occurs in the course of many different acute febrile affections, and as one of their sequelæ.

2. *At other times the coagulation of the diseased mass of the blood within the tube of some one vein is the primary, and indeed the special occurrence, which gives rise,* from reaction as it were, *to inflammation of the coats of the veins—phlebitis. This disease is then a dependent, secondary affection, of subordinate importance.* Such a form of phlebitis always consists in the establishment of a disease of the blood, which is either of a spontaneous character, or depending upon the absorption of different deleterious substances, such as inflammatory products originating either within or external to the vascular system. It constitutes the most frequent of what are termed metastases, especially if we include the process of the coagulation of blood in the capillaries—the so-called *capillary phlebitis.*

This distinction of character, which has hitherto not been sufficiently regarded or properly understood, is of the greatest practical importance and interest, since it affords a clue to the right comprehension of the significance of phlebitis in individual cases, and thus contributes to throw light on many points in the history of phlebitis which had either remained entirely obscure, or had been explained in a wholly irrational manner. We shall always indicate this latter form of phlebitis as that which depends *upon coagulation of the blood.*

A. The following are the anatomical indications of (acute) phlebitis:

1. *Injection and Redness of the Cellular Coat of the Vein,* in different degrees and shades of color. The cellular coat is very commonly intersected by varicose vessels, and is at the same time of a bluish-red color, which, however, experiences various modifications by the infiltration of inflammatory products into its tissue. The latter membrane very frequently presents a darkish-red, mottled or streaked appearance, in consequence of slight extravasations.

2. *Infiltration of the Cellular Coat,* with a serous, sero-fibrinous, partially solidifying, grayish, gray or yellowish-red moisture, and bulging of the coat; the infiltration is very commonly associated with a thin sero-purulent, or thick purulent moisture and with bulging, whilst more or less circumscribed abscesses occur in the interior of the vessel.

The neighboring cellular tissue participates, in various degrees, in the process, although, generally speaking, in proportion to its vicinity to the seat of the disease; the tissue becomes vascularized, infiltrated, and swollen, and the vein becomes then fixed or imbedded in it. We also very often observe circumscribed abscesses, together with diffused purulent infiltration at some distance from the vein.

3. *Injection and Redness,* as well as *the other discolorations* produced by the infiltration of various products, *extend into the circular fibrous coat of the vein.* The latter coat appears to be vascularized; but more frequently the injection and redness of the tissue are already obliterated. It then presents a grayish-yellow faded appearance, and is discolored at different points by imbibition from within, or from the contiguous extravasations in the cellular coat, or is mottled red by small extravasations within its own tissue; it is, moreover, unusually succulent, and is swollen. In phlebitis with purulent exudation, it is most distinctly infiltrated with the purulent or sero-purulent fluid.

4. *The inner coat of the vessel presents a dirty-white appearance, or*

is colored red, violet, brown, or even green, by the imbibition of hæmatin from its interior, or from hæmorrhagic exudations into its tissue. It is swollen; the inner surface is devoid of lustre, is dull, felt-like, and wrinkled. When purulent exudation is present, it is more especially of a pale yellowish color, succulent and lustreless.

5. In addition to these alterations, *all the coats of the vein are relaxed in their texture, and admit very readily of being torn, and separated from one another.* In some cases the strata composing the inner coat (together with the valves) are detached from the circular fibrous coat, and even cast off in the form of a tube; and this is of very general occurrence in the more intense forms of phlebitis with purulent exudation.—In these cases, the inner coat, which is thrown off in the form of a tube, may be the more readily mistaken on a superficial examination for a tubular exudation, when its tissue admits of being readily torn, and it has been colored yellow by the imbibition of pus.

6. *The vein appears to be dilated and paralyzed; its tube is generally either filled by a plug of blood*, which either resembles a recent coagulum, or has entered into different metamorphoses, *or is filled with the product of the process* (*the exudation*), *more especially pus.* The formation and significance of the above-named coagulum are intimately connected with the actual process of exudation, as will be seen by the following remarks:

7. *Exudation.*—The exudations deposited in the texture of the venous coats, and in the contiguous tissues surrounding the vein (its cellular bed), have already been in part considered, both in reference to their bulk and nature. A far more important class of exudations are *those which are deposited on the inner free surface of the vein* (*within the vein*), *and which, owing to their absorption into the blood, and the infection to which they may give rise*, impart to phlebitis the dangerous character that renders it so formidable a disease. The general disease arising from these conditions, together with its intensity and character, depends upon the nature of the exudation, and also upon certain accidental circumstances, which we now proceed to consider.

a. The exudation may vary very considerably, both in reference to its physical properties and its internal composition. A direct anatomical demonstration of the exudation itself, either in reference to the originally inconsiderable quantity in which it appears, to its absorption into the blood, or still more in regard to the evidence of its original and special quality, is very difficult or even impossible. We would especially notice:

The so-called plastic exudation, capable of undergoing a metamorphosis of tissue, which, in rare cases, occurs in an appreciable quantity as a flocculent, soft, or consistent membranous coagulum on the inner surface of the vessel, or adhering to the fibrinous plug which fills the vein. The quantity of exuded serum originally contained within it never admits of being detected, as it is absorbed by the blood at the moment of its exudation, together with the greater portion of its coagulable matter.

The purulent and ichorous exudation, which is a very frequent product of phlebitis, is generally secreted in such abundance, that it may be easily recognized, even in those cases in which the blood has coagulated within the vessel. It very commonly expels the blood entirely from the vein, which is then completely filled with pure pus or ichor, the product of the process of exudation. We, moreover, here meet with exudations of a fibrinous, purulent product deposited under the lining membrane of the vessel, in the form of islands or large patches, exhibiting diffused, purulent, ichorous infiltration of the venous coats, together with discoloration, loosening, and a high degree of lacerability and detachment of the strata composing the lining membrane, in the form of a lax, lacerable, disintegrating fusing cylinder, which might easily be mistaken for a tubular exudation.

Are any of these exudations of a hæmorrhagic or a tuberculous character? In many instances, indeed, we observe a red, brownish or violet-red, or chocolate-brown coloration of the exudation on the inner surface of the vessel, together with a hæmorrhagic suffusion of the coats of the vein, and centres of hæmorrhagic exudations in the neighborhood of the vessel.

We have never observed a tuberculizing exudation, or even one whose nature led us to suspect a tuberculous character, on the inner surface of the vein. (See our subsequent remarks on Tuberculosis.)

b. The coagulation of the blood in the inflamed vessel, or the formation of a fibrinous plug, which is most intimately connected with the deposition of an exudation upon the inner surface of the vein, and with its absorption into the blood, is a phenomenon of the greatest and most varied interest. It arises from the contact of the blood with the products of inflammation. The subject has been already generally considered under Diseases of the Blood, and will therefore be noticed here only in as far as is indispensably necessary towards the right comprehension of phlebitis.

Phlebitis, if we may judge from appearances after death, very rarely occurs without a simultaneous coagulation of the blood in the inflamed vein.

The inflamed vein is very commonly filled by a cylindrical fibrinous plug, which, according to circumstances, is either single or ramified, and terminates conically at both extremities.

In phlebitis having a purulent exudation, the coagulum is either present in the above-described form, or is disintegrated, and blended with the purulent product in the form of loose, friable detritus; or, finally, there may be no trace of its presence, in which case the vein is entirely filled by copiously exuded pure pus.

8. Further evidence of the phlebitic process, both in reference to its own nature and that of its products, and to its highly important character, is afforded by numerous secondary conditions, which we shall briefly notice in the following remarks, referring our readers to our previous observations on the subject in Diseases of the Blood.

a. The immediate consequence of the phlebitic process is a *diseased condition of the blood*, arising from the absorption of the morbid products, which constitutes the basis of all the subsequent secondary phenomena. This disease generally induces degeneration of the blood, according to the character of the product, either into a so-called phlogistic condition (hæmitis, hyperinosis), or into pyæmia. Hence arise:

b. The processes of stasis and coagulation of the blood in various portions of the capillary system (lobular processes, deposits, metastases, capillary phlebitis), with the different metamorphoses of such a coagulation; namely, shrivelling to a fibroid callus with atrophy of the tissue, or purulent, ichorous fusion with similar destruction, necrosis of the tissue; *processes of coagulation in larger vessels, more especially the veins;* and finally in the *heart itself*, under the form of different vegetations.

c. The allied processes of stasis and of exudation into the parenchymatous structures, as well as upon the membranous, serous, and mucous surfaces, with the fusion of the substratum, which is especially perceptible on the mucous membranes, and with suppuration and necrosis of the tissue.

This general infection of the blood by the product of phlebitis, together with the further phenomena depending upon that process, does not, however, invariably take place,—a circumstance that some observers attempt to explain, in imitation of Cruveilhier, by the so-called *sequestration of the vein.* Thus, for instance, the coagulation of the blood on the limits of the inflammation, and the exudation into the vein, are supposed to isolate the inflammatory product—the pus—and prevent its absorption into the blood.

We have been led, by extensive experience, to adopt the following views in reference to the solution of these two intimately associated questions regarding the cause of the non-occurrence of a general infection, and the significance of the so-called sequestration, as a special means of arresting the process.

The non-occurrence of a general infection of the blood in phlebitis would appear to be frequent, if we judge from observations on the living subject; but, on the other hand, it is rarely noticed after death, where the phlebitis which is brought under our observation is generally characterized by purulent exudation.

In the latter cases, therefore, the exudation must be absorbed into the blood, and carried away with it from the seat of its formation.

The reason of the non-occurrence of a general infection depends, in our opinion, upon the fact of the blood coagulating at the place of the exudation, and upon the rapidity with which the whole of the blood, or one of its strata, is coagulated in consequence of the absorption of the inflammatory product; whence the course of the recently deposited or still exuding product is at once arrested.

As, however, this coagulation in the ordinary and more frequent cases is not effected immediately, but requires (as we see exemplified in the frequent development of coagula in a section of the vascular system remote from the infected portion of the blood) that the heterogeneous substance must remain for some time in contact with the blood, we are able to explain why a portion of the exudation is in general carried onwards by the circulation, and the blood is then infected before the coagulation can be established in the vessel.

A sequestrating fibrinous plug must be distinguished from the *coagulum originally filling the vessel, and induced in the blood-current by the absorption of the exudation.*

It is certainly true, that in every case of phlebitis the coagulation of the blood extends beyond the limits of the inflammatory centre, along the vessel, and the coagulum filling it. In order to comprehend the significance of this coagulation as a means of sequestration, it will be necessary for us to form a clear idea of the conditions requisite for its formation.

The coagulation is effected in a simple manner, above and below the inflamed vein, and around the coagulum which originally filled it. The blood is coagulated below the inflamed vein (at its circumference) in all the branches where it is retained by the coagulum obstructing the vessel; above this point, towards the centre, the blood is arrested as far as the next considerable-sized venous branch that opens into the diseased vein. The coagulation is thus dependent on the coagulum originally obstructing the inflamed vein, and is essentially a thrombus.

Seeing that this form of coagulation can only be effected after the formation of the original coagulum, there are two points to be considered in reference to its significance.

a. A general infection usually occurs, as has been already stated, before the development of the coagulum which originates in the absorption of the exudation, and consequently still longer before the formation of the sequestrating clot.

b. The original coagulum is rapidly formed after the deposition of the exudation, and it hinders the general infection by entirely filling up the tube of the vessel, and absorbing the whole of the exudation. The sequestrating plug does not appear, in either case, to be of any essential service.

We here, however, draw the following inferences in reference to the possibility of infection arising from the metamorphosis of the original coagulum:

1. If the phlebitis had deposited a so-called plastic exudation, and belonged to the form which terminates in disintegration or obliteration (see the modes of termination of Phlebitis), the sequestrating plug would be of no obvious utility in either of the cases considered under *a* and *b*.

2. If the phlebitis had deposited a purulent ichorous exudation, there would necessarily have existed one or other of the following conditions:

a. A general infection of the blood must have been induced previously to the development of the original coagulum, which may either have filled the diseased vessel throughout its entire length, or may have been limited to the margins of an accumulation of pus in the vein, if that fluid were exuded in large quantity. The sequestrating plug cannot, in such a case, hinder the pyæmia, in the course of whose existence it has, in fact, been developed; while it is itself, moreover, subject to purulent fusion from a prolonged continuance of the disease.

b. Or the original coagulum may have been rapidly formed by coming in contact with the pus, in which case general infection of the blood could not possibly have been effected at that moment, but inasmuch as this coagulum undergoes a more or less rapid purulent fusion, there is a possibility of the blood becoming secondarily infected by the disintegrated admixture of the coagulum. This coagulum may be permanently retained through a subsequent coagulation of the blood that has continued unaffected. It is only under these conditions that the coagulation which occurs at the limits of the inflamed vein is of essential use—that is to say, *it is only the pus proceeding from the metamorphosis of a coagulum established in the vein by the absorption of a purulent exudation produced at the spot, that can in the true sense of the word be sequestrated.*

In addition to the signs and consequences of phlebitis to which we have already referred, there are certain *associated and consecutive phenomena*, which still require notice; namely, accumulation of blood in the small veins and capillaries beneath the inflamed vein, a cyanotic tint, and œdema around this portion of the vascular system; these are consequences of the occlusion of the inflamed vein. As the inflammatory process extends, we have inflammation of the cellular sheath of the vein and of the surrounding cellular tissue, with sero-fibrinous, sero-purulent, hemorrhagic exudations; we further have inflammation of the skin in the form of erysipelatous redness, which, from the beginning, accompanies the inflammation of the subcutaneous veins in the form of red streaks along the course of these veins; and, as a final result, we have moist gangrene, caused by the stasis established in the capillaries by the extensive occlusion of the inflamed vein.

The following are the *terminations of phlebitis:* it may end in resolution; in chronic inflammation, with persistent thickening; in coalescence of the vein with contiguous structures; and in dilatation, in persistent obliteration, or in suppuration of the vein.

1. The *termination in resolution* (or perfect recovery) is by no means rare in slight cases of phlebitis, even where there has been a general infection, like that occurring in endocarditis, which, independently of the local residua, very frequently assumes a similar favorable character, notwithstanding the pre-existence of general infection. The coagulum obstructing the inflamed vein is gradually absorbed into the blood in a finely comminuted state, and being dissolved like the originally absorbed exuded substance, the diseased vein becomes again free.

2. In some cases there remains *a condition of chronic vascularity of the coats of the vein, with bulging*, which is not unfrequently accompanied with a rusty-brown or slate-gray discoloration, with paralysis and dilatation of the vein. In this condition there are often acute relapses, more especially in the veins of the lower extremities. It finally leads to hypertrophy of the coats of the vein, in consequence of the continued accumulation of the exudation in their tissue; to rigidity of these coats; to permanent dilatation, and by means of the sclerosis of the surrounding cellular tissue, to permanent immobility of the vein, and coalescence with the neighboring structures—as, for instance, aponeuroses, muscular sheaths, the general investments, the periosteum, &c. The latter condition further predisposes to the formation of new layers of lining membrane from the blood, in consequence of the retarded flow of the blood-current.

3. *Termination in obliteration* is induced by means of the coagulum, which obstructs the inflamed vein. This coagulum, which is formed by the absorption of a so-called plastic exudation (that is to say, an exuda-

tion capable of being metamorphosed into tissue), undergoes a gradual decoloration and is converted into a whitish fibroid band, which is very commonly interspersed with rusty-brown or black pigment. After this metamorphosis, the coagulum becomes shrivelled.

This string or band is attached to the wall of the vein, either at all parts of the circumference, or only at separate points, by means of a cellular structure, formed by the metamorphosis of a portion of the exudation on the inner surface of the vein. If the wall of the vein participate, in the former case, in the shrivelling of the coagulum, the vessel is rendered impermeable, and becomes finally atrophied, and *completely obliterated.* But if, on the other hand, the wall of the vein does not generally participate in the shrivelling of the coagulum, in consequence of the latter being attached merely by partial adhesions, or if the blood forces its way into the obstructed vein notwithstanding the total adhesion of the coagulum, the structure by which the shrivelling coagulum is attached to the wall of the vein becomes torn into threads and laminæ, which gradually acquire an investment of recently formed lining membrane from the blood; and the vein being in consequence only partially obliterated exhibits the following appearances:

The vein is either occupied by a fibroid, roundish string, which adhering only to a portion of the wall of the vessel leaves the latter free and permeable in other parts.

Or, in addition to this connection of the fibroid string with the vessel, thread-like bridges, or membranous partitions, which are more or less perforated—the torn adhesions above referred to—are also attached to the free portion of the wall of the vein, while its tube is broken up into numerous straight or oblique canals or divisions.

Or the fibroid string is attached at various parts to the interior of the vessel, by means of adhesions, arranged in the most irregular manner.

The coats of the vein are thickened, the vessel itself being more or less firmly imbedded in a cellular stratum in a state of sclerosis; the free portion of the wall is dilated and elongated, so that the vessel describes intestine-like coils round the resistant fibroid string, or twists itself around it as around an axis, as the former changes the points of its adhesion. This condition gives rise to a special form of varicosity. The vessel at the same time presents some analogy in its calibre with the structure of the sinuses of the dura mater, more especially with the superior longitudinal sinus.

This fibroid string within the vein may ossify in the progress of time, constituting a form of central ossification.

This condition has been more especially observed in the cutaneous veins of the lower extremities—on the trunk and ramifications of the saphena veins. We noticed it in one case, together with the products of intense peritoneal inflammation, on the whole system of the mesenteric veins; and on account of its rarity, we will give a brief history of it.

The body of a girl, aged 13 years, exhibited the following appearances, in addition to an excessive degree of emaciation, and a pale decoloration of the general investments. The abdomen was swollen, and felt hard and board-like to the touch; the linea alba presented the resistance and the appearance of a cartilaginous layer, and was a line and a half in thickness; the same character was generally exhibited by the aponeurotic portions of the abdominal wall. The peritoneum of the abdominal wall was invested by a pale slate-gray, cartilaginously tough pseudo-membrane, a quarter of a line in thickness, which extended to the intestinal canal and the stomach, which it covered. It contained a yellowish-white purulent fluid. The mesenteric vein, with its ramifications, was partly thickened and callous, while its canal was divided by numerous bridge-like partitions, which were perforated at different spots, and whose margins were torn, and was partly contracted by detached structures of this kind. The walls of the portal vein were uniformly thickened. The coats of the hepatic, cystic, and common bile-ducts were swollen, whilst their mucous membrane was covered with numerous, and generally suppurating, villous growths; the ducts contained an ichorous, brownish fluid. A similarly colored dark fluid was also found in the stomach and the intestinal canal. The patient, in addition to general indisposition, had suffered from chocolate-colored discharges, both by stool and vomiting, and had also, throughout her illness, vomited light-red, pure blood, which proceeded from the gall-ducts.

Cheesy matter, and a substance resembling moist chalk or mortar, are, moreover, occasionally found in veins that have been obliterated, and are either the disintegrated remains of the coagulum which had obstructed the vessel, or inspissated pus.

4. *The termination in suppuration*, and in an acute purulent fusion and necrosis of the coats of the veins, corresponding to the previous (acute) course of the disease, is, on the whole, somewhat rare. The dilated vein is filled with a large quantity of purulent exudation, mixed with blood, and all its coats, besides presenting a dirty-red tint from imbibition, are infiltrated with pus, and tear with great facility and almost like tinder; while the inner coats peel off from the cellular sheath in the form of a crumbling pipe or tube, and the cellular tissue surrounding the vein is in a state of suppuration. This process sometimes occurs at individual spots, so that, after the solution of the venous wall, circumscribed abscesses are occasioned in the adjacent cellular tissue. This acute suppuration of a vein usually only occurs when the highest degree of pyæmia has been developed. A more frequent occurrence is a protracted suppuration in sequestrated varicose veins, which had originally suffered from chronic inflammation, a subject to which we shall again recur. The phlebitis induced by the coagulation of blood often terminates in *gangrenous suppuration and fusion.*

From what has been already stated, both in reference to the exudation and the last two modes of termination, it follows that the phlebitis of which we have already treated is sometimes *adhesive* and sometimes *suppurative.*

B. *The phlebitis depending on coagulation of the blood* differs from the form of acute phlebitis hitherto treated of, inasmuch as the coagulation within the vessel is the primary phenomenon, whilst the inflammation of the coats of the veins—phlebitis—is associated with it merely as a secondary affection. The coagulation is therefore not occasioned by the inflammatory product of the coats of the veins—that is to say, by the absorption of the exudation into the blood from the inner surface of the vein, but is the result of a disease of the blood, which is either spontaneous or occasioned by the absorption of different products of stasis or inflammation, deposited either within or external to the vascular system. This disease reaches so high a degree of development at different points, and in the second case at different distances from the centres of infection, that the column of the blood coagulates more or less rapidly, with a more or less complete separation of the fibrin. When the coagulum is once formed, inflammation of the coats of the veins, if not invariably and very rapidly developed, is at all events of very common occurrence.

The existence of this process, when developed in the manner above described, proves the undoubted occurrence of coagulation of the blood in the various portions of the vascular system, from the centre to the capillaries, even where there is no trace of inflammation in the vessel; but it does not prove that where inflammation of the vein is present, its intensity and development have been sufficient to cause the coagulation by the deposition of exudation on the inner surface of the vessel.

The indications of this phlebitis are, in general, identical with those observed in the inflammation of the veins which we have already considered, but it is nevertheless of the greatest importance to notice the following special points:

1. We very frequently observe the above indicated want of relation between the nature and metamorphosis of the coagulation and the degree of intensity we should expect to meet with at the beginning of true inflammation of the coats of the veins. The disease generally, however, exhibits a very slight intensity, while the lining membrane of the vessel is not in a condition which would seem to indicate the immediate pre-occurrence of an exudation into its tissue, extending by means of the latter to the inner surface of the vessel. This is the most remarkable, since, as we shall see by the following facts, it is usually owing to a purulent exudation that the process is developed in its subsequent course.

2. In general it is an ordinary pyæmia which occasions the coagulation of the blood in vessels of considerable calibre. In accordance with this view, the coagulum commonly undergoes a purulent metamorphosis, a fusion into a more or less organized pus or ichor. According to the circumstances of the case, the vein finally contains a chocolate-brown, grayish-red, or yeast-yellow purulent fluid, mixed with partially dissolved fragments of the plug; or a dirty-brown, brownish-green, fœtid, ichorous fluid, or even a very discolored, stinking, gangrenous ichor (Phlebitis septica). The contents are here not the product of the inflamed venous wall, but proceed from the metamorphosis of the coagulum.

3. In consequence of this metamorphosis—that is to say, of the contact of the inner coat of the vessel with the deleterious substance and its subsequent imbibition, the inflammation of the coats of the veins rapidly attains a high degree of development, and gives rise to corresponding purulent and ichorous exudations, which are added to the above described contents of the vein.

4. The diseased condition of the blood is in a high degree the con-

trolling cause, while the so-called metastatic processes, to which it gives rise, are distinguished by their number and intensity.

5. An isolating or sequestrating coagulation cannot, as is obvious, be in any way conducive to this process.

It must be remarked, in reference to *the terminations of this phlebitis*, that—

1. The ordinary termination, in case death be not sooner induced by the general disease, is *an acute ulcerous fusion, a gangrenous and ichorous destruction of the vein*, arising from the process already considered under 2.

2. It is very rare, in accordance with the facts above referred to, for the disease to terminate in *permanent occlusion, or in complete or incomplete obliteration.* When the pre-existing inflammation of the coats of the veins has attained so high a degree of development as to cause a plastic exudation to be deposited on the inner surface, it will give rise to the adhesion of the obstructing plug; this may also be effected by the direct coalescence of the lining coat of the vessel with the plug.

To this form of phlebitis belong also those processes in the capillary system of the different tissues which have been commonly designated lobular processes, metastasis, and capillary phlebitis by the French. They are, in fact, the same process which we have already considered as that form of phlebitis which is induced by coagulation of the blood. We shall revert to this subject when we enter upon the consideration of the diseases of the smaller vessels, and of the true capillaries.

Although we think we need hardly enter upon any special discussion of the differential diagnosis of these two forms of acute phlebitis, after having considered them with every possible attention under the heads A and B, we would yet draw attention to the following additional remarks:

The division of phlebitis into an *adhesive* and a *suppurative* form is well known, and has been generally followed. The two forms we have established may participate in either of the above characters. We would, however, expressly notice the error into which French observers have fallen, in regarding the purulent mass, which is in the centre of the coagulum, as the product of the inflamed coats of the veins. Cruveilhier attempts to show that the pus reaches the coagulum from without, by the capillary action of the coagulum; but this very unnatural hypothesis is quite inadequate to the solution of the question. We know, from many highly important analogies, that in ordinary cases the metamorphosis of the coagulum begins at its central nucleus, and that the purulent matter in the midst of the plug obstructing the vein cannot be regarded as the product of the inflamed coats of the vein.

C. *Chronic Phlebitis*, as already observed at p. 256, is occasionally a consequence of the acute form of the disease; it may, however, likewise occur independently of the latter.

In the latter case it consists in chronic inflammation of the cellular coat, into whose tissue, and the contiguous layers of the circular fibrous coat, it deposits its products.

The cellular substratum of the vein must obviously participate in this process. Its anatomical indications are chronic inflammation of the cellular tissue.

It gives rise to dilatation, varicosity, and thickening, through the hypertrophy and sclerosis of the cellular coat of the vein; and, secondarily, through the new layers of lining membrane that are formed from the impeded blood-current, to the gluing of the vein into its cellular bed, to rigidity of the venous coats, and to gaping of the cut tubes; in short, it makes the veins approximate in character to arteries.

It has a great tendency, on very slight provocation, to pass into acute phlebitis.

Its most obvious causes are persistent distension and dilatation of the veins, in consequence of the impediments presented to the passage of the blood through them, besides which, it very often arises from the inflammation of the contiguous cellular tissue by an extension of the process. In accordance with what has been already stated, this condition is especially frequent in varicose veins, and in the veins of the lower extremities, where it very frequently originates in the subcutaneous tissue, which is then the seat of chronic inflammation, arising from habitual eczema.

Phlebitis very generally follows the course of the blood-current towards the heart, at all events during the early stages of its development; but yet exceptional cases are not unfrequently noticed, in which the inflammation follows a different course, and extends, during its more advanced stages, in an opposite direction.

It still remains for us to notice the phlebitis which attacks some special portions of the venous system.

1. *Inflammation of the sinuses of the dura mater* arises from injuries of the cranium, in consequence of a concussion of the dura mater near the sinus, or of a direct injury of the latter from fragments of bones, &c. It is frequently observed to arise from inflammation of the dura mater in the neighborhood of a sinus, and from inflammation and suppuration of the bone. A tolerably frequent example of the latter mode of derivation presents itself in the inflammation of the sigmoid sinus,[1] arising from caries of the petrous portion of the temporal bone, whence it commonly spreads, with considerable rapidity, to the internal jugular vein. In some rare cases, this form of phlebitis is owing to coagulation of the blood (metastasis); and as it is almost invariably accompanied, under these conditions, by a purulent exudation, we not unfrequently find that the walls of the sinus exhibit incipient suppuration. We have repeatedly seen cases in which the inflammation, after attacking the cavernous sinus, had extended by means of the superior ophthalmic to the anterior facial vein, and exhibited a diffused redness over the skin of the face.

In inflammation of the longitudinal sinus, the venous trunks opening into it are occluded at the convexity of the hemispheres.

2. *The last-named vessels* occasionally become the seat of coagulation, in consequence of disease of the blood, which is either spontaneous, or induced by the absorption of pus. This coagulation, in its turn, gives rise to phlebitis, which, together with the inflammation of the sinus, is frequently associated with meningitis.

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[1] [The anterior portion of the lateral sinus.]

3. *Inflammation of the trunk and branches of the portal vein in the liver.—Inflammation of the portal branches within the liver, and their adhesive inflammation, resulting in obliteration*, are not of uncommon occurrence, as will be seen from the following remarks:

We not unfrequently find, at different spots within the liver, more or less widely diffused accumulations of a callous (cellulo-fibrous) tissue, generally of an irregular, ramifying form, and, as a careful examination shows us, following an obliterated branch of the portal vein. In the obliterated vessel we sometimes find a yellow, cheesy, chalky, or mortar-like inspissated plug—the remains of a fibrinous coagulum, closing the inflamed vein. When these callosities are of considerable size and number, and especially when they are on or near the surface of the liver, this organ undergoes a striking change of form, and presents a lobulated appearance. As a consequence of the obliteration of a branch of the portal vein, the surrounding parenchyma becomes atrophied into these cellulo-fibrous masses, which subsequently shrivel by contracting, exert tension on the contiguous tissue, and, if they reach the surface of the liver, on its peritoneal coat; so that in this case they produce cicatrix-like depressions and furrows on the surface, which give to the still unaffected part of the liver a flattish, round, lobulated appearance. This form of inflammation seems, in most cases, to be secondary, and to be phlebitis induced by the coagulation of blood.

Inflammation of the trunk of the portal vein, and of its principal branches, with purulent infiltration, is undoubtedly of very much rarer occurrence. We have repeatedly observed it with incipient ichorous destruction, and necrosis of the coats of the vessel. It invariably gives rise to innumerable purulent and ichorous abscesses in the liver, dependent on capillary phlebitis; and also to such abscesses in the lungs, with a very highly developed pyæmia. Lambron has observed a case of pylephlebitis, which was produced by the penetration of a fish-bone through the pylorus and the pancreas into the trunk of the superior mesenteric vein. (Arch. gén., Juin, 1842.)

4. *Inflammation of the uterine veins, especially after delivery*, is the most common form of this affection. It attacks the gaping venous sinuses at the points where they are torn from their insertion into the placenta, and extends from thence through the veins and their plexuses which run through the substance of the uterus towards its lateral walls. From hence it very often extends to the plexus pampiniformis and the trunk of the internal spermatic vein, and finally even to the trunk of the vena cava; or occasionally (but much more rarely) to the hypogastric (internal iliac) vein, and from thence to the veins of one or both of the lower extremities (Phlegmasia alba).

It usually occurs as a *substantive inflammation of the coats* of the veins, which commences from their insertion into the placenta, constitutes an integral constituent of the exudative process on the inner surface of the uterus, and extends in the direction which we have already indicated. It usually deposits purulent, ichorous, or septic products, corresponding to the product of the uterine exudative process. It is, however, not unfrequently *induced by a coagulation of blood* at various points of the uterine veins, in the spermatic vein, the vena cava, or in the most various and remote portions of the venous system. The exudation produced on the inner surface of the uterus, or an exudation deposited by substantive phlebitis of the uterine veins near their insertion into the placenta, becomes retained in the blood, and occasions its coagulation, either at once in the immediate vicinity, or subsequently at varying distances from the centre of infection, after a more prolonged action on the blood.

The pyæmia developed after delivery is one of the most frequent and intense forms of this affection.

5. *Inflammation of the umbilical veins of newborn infants*—a phenomenon somewhat frequently combined with ulceration of the navel, and accompanied by erysipelatous redness of the skin of the abdomen, very often with jaundice, and not unfrequently with peritonitis—never, on the other hand, is followed by secondary processes of capillary phlebitis. In this respect Duplay's opinion coincides with my own. Its products, therefore, in consequence of this circumstance, do not produce any infection of the blood, which is doubtless owing to the fact, that no circulation takes place through this vein after birth.

6. *Inflammation of the vena cava ascendens* is induced in puerperal women by the coagulation of blood, extending from the inner spermatic vein into the vena cava. Under other conditions, it also appears to arise from a similar cause.

In addition to these special cases we must mention:

α. *Phlebitis from a wound complicated with the introduction of a deleterious substance.* The wound, besides involving other structures, affects either a large vein or only capillaries. In the former case, the phlebitis following the wound is *either* the consequence of intense inflammation and of low suppuration in the soft parts, and in the wounded vein,—the general symptoms, if they are present, being not produced by the direct absorption of poisonous matter into the wound from without, but by the products of inflammation of the injured structures, and especially of the veins; *or* the case may be altogether different; the poisonous matter may penetrate into the vein when it is opened, and be taken up into the blood,—the general symptoms and the poisoning of the mass of the blood being here developed rapidly. According to the nature of the substance, there may or may not be a coagulation in the wounded vessel; when there is a coagulation, we have a further inflammation of the venous coats.

In a wound affecting the capillaries, the poisonous substance, either in a state of purity, or mixed with the products of consecutive inflammation and low suppuration, enters the vessels either directly through their open mouths, or by imbibition through their walls; the phenomena are then either general, or, in accordance with the quality of the substance, we have a coagulum in the vein, either near or at some distance from the wound, and inflammation extending from it.

β. *Cancerous Phlebitis* especially occurs in the uterine veins, from whence it extends into the internal spermatic, the hypogastric (internal iliac), and the femoral veins, in cases of cancer of the uterus. These veins are closed by a coagulum of cancer, especially of the encephaloid form, in various stages of metamorphosis. This coagulum finds its way into the venous blood by the absorption of the cancerous matter in various modes, either as blastema, or cancer-cells, or cancerous ichor. Hence this condition, when it actually occurs as phlebitis, is that form which is induced by a coagulation of blood and its metamorphosis.

b. Hypertrophy of the venous coats, especially of the lining membrane.—We shall treat of this subject in the present place with the diseases of texture as in the case of the arteries, and shall allow it to follow phlebitis, because on the one hand, hypertrophy of the venous coats is intimately allied to inflammation of the veins, and because, on the other, without a previous explanation of the mode in which an excessive formation of the lining membrane takes place within the vein, many points in the following pages would hardly be intelligible.

Hypertrophy of the whole venous wall especially consists, on the one hand, in an augmentation of the mass, and in a simultaneous sclerosis of the cellular coat of the vein, and, on the other hand, in a thickening of the lining membrane of the vessel; an augmentation in the bulk of the circular fibrous coat is generally less marked; it is caused by the persistent impediment to the blood-current, and by the accumulation of blood in the vein, while the increased bulk of the cellular coat is principally occasioned by chronic inflammation of the vein, which is commonly perceptibly dilated, and at the same time assumes, as we have already remarked, a sort of arterial habitus.

Thickening of the inner coat of the vein is the especial result of an irregular formation of new layers from the blood; it is an endogenous production. Its occurrence in the venous system is rare, as compared with its frequency in the arteries, nor do we meet with it in the same highly developed form; moreover, in the veins it never occurs as a general constitutional disease, but is merely deposited through local conditions, such as the impediment presented to the current of blood, and occasionally the entrance of arterial blood into the vein.

The form under which the deposition of new layers of the lining membrane appears in the vein, is principally—

a. The same as that in which it generally occurs in the arteries; it usually constitutes an opaque, white, smooth, and plane stratum, admitting of being cleft into more or less numerous lamellæ; occasionally it forms a thickish, nodular, and uneven layer; and sometimes it presents a reticulated or areolar appearance. The vein never attains that considerable degree of thickness which is so frequently observed in the arteries. In reference to its metamorphoses, we very rarely observe an ossification; in veins which lie by ossifying arteries, and are fixed in a bed of inflamed cellular tissue, as, for instance, the femoral veins, we observe the process of ossification chiefly occurring in the wall of the vein nearest to the artery.

b. As a second form of these anomalies, and one which is altogether peculiar to the veins, we must notice the *vein-stones* (*phlebolites*), which have in general not been sufficiently regarded by writers on diseases of the veins. We believe that we have recognized their true nature, and that they deserve to be considered in the present place. They are concretions of a round, oval, or cylindrical form, commonly of the size of a hemp-seed, a pea, a bean, or even of a hazel-nut, and of a white or whitish-yellow color, which either lie free (as is most commonly the case) in the vein, or, if they are of more considerable size, become wedged and fixed in the vein, and entirely close it; or, finally, they adhere to, or actually coalesce with, the lining membrane of the vein, by means of a cylindrical or fusiform projecting coagulum, or through delicate membranous structures. Large phlebolites often lie in saccular pouches on the side of the vein. In some cases, this pouch, together with the phlebolite, separates from the vein, when it and the vein are closely compressed in a capsule, formed from the wall of the vessel. The vein then exhibits a more or less distinctly contracting cicatrix at the spot. When the lining and the circular fibrous coats of the capsule are gradually destroyed, the phlebolite finally lies in a capsule of cellular tissue, and this appearance may have given rise to the opinion that the phlebolite is originally developed in the cellular tissue outside the vein. Phlebolites are of very common occurrence, either separately or in small groups, more especially in the pelvic veins; that is to say, in the vessels of the bladder, vagina, uterus, and rectum of old persons; they also occasionally appear in other portions of the venous system, and even occur in young persons. They are also sometimes met with in the spleen, and in the form of sand-like concretions in the cellular spaces of certain teleangiectases (as for instance supplementary spleens).

A careful examination of phlebolites, and an attentive consideration of the circumstances under which they are formed, yield the following results:

On cutting through the phlebolite, we discover that it is of a concentrically stratified structure; that the innermost lamellæ are usually of a whitish-yellow color, and the outer ones white, the former being compact and exhibiting a glass-like brittleness, whilst the latter are softer and of an earthy texture. The outermost layers are composed of soft membranes, are usually white and opaque, and exhibit at different points a gelatinous translucence. The membranous structures, by means of which phlebolites are sometimes attached to the inner wall of the vessel, are prolongations, or duplicatures of this wall, and, as it were, coatings of the peripheral layer investing the concretion. There is very commonly a roundish cavity, or, instead of it, an irregular fissure within the nucleus of the phlebolite, which is dry, and of a rusty brown, or dull yellow color. The surface of phlebolites, in some rare cases, exhibits the appearance of being gnawed at separate points to different depths; and these spots are occasionally invested with a faded yellow, fatty, soft mass. The chemical analysis of phlebolites shows that they consist of an animal substratum, with phosphate and carbonate of lime and some magnesia. (John, Gmelin, Lehmann, and Hasse.)

The conditions giving rise to the formation of phlebolites are, as far as we know, a retarded flow of the blood in dilated veins; thus we observe them in the pelvic veins of aged persons, and in these and other veins in young persons, in consequence of an impediment to the circulation of the blood by the pressure of fibrous tumors of the uterus, enlarged ovaries, or prolapsus of the uterus in women, or of an enlarged prostate gland or distended bladder in men. We have seen a case where the subcutaneous veins on the abdomen had become varicose, in consequence of anastomosing with the umbilical vein, and of its remaining patent, and were so plugged up with phlebolites, that the skin felt as if it were full of shot.

We are of opinion that there is first a slight coagulation of blood in the vein, around which, there is deposited in concentric layers a structure analogous to the lining membrane of the vessel and the deposition in the arteries, and formed from the plasma of the blood. These strata generally become opaque, and ossify from the interior towards the circumference in much the same order as that in which they have been produced, or in some comparatively rare cases, they undergo the atheromatous process. The nucleus which is formed from the coagulum then shrivels, assumes a rusty brown or dull yellow color as it dries, and leaves a cavity in the centre of the phlebolite varying according to its previous volume; or it undergoes cretefaction, and cannot be recognized within its calcareous capsule.

The large coagula which occur together with phlebolites in varicose veins, cannot be regarded, at least when of the ordinary size, as the first step in the formation of phlebolites. They are observed in all varicose veins, including even those in which phlebolites are of rare occurrence; nor do they always present a concentrically stratified structure. It may, however, very probably be the residua of these strata, remaining after their general solution, which furnish the nucleus for a subsequent phlebolite.

The view which we have advanced in reference to the disease of the veins considered in the present section, makes it worthy of notice, that, in addition to the differences it presents when affecting the veins instead of the arteries, this disease of the veins is characterized by the extremely rare manifestation of the atheromatous process in either of the forms of deposition from venous blood; indeed, as far as we are aware, it never occurs in a deposit of the first form, notwithstanding its great frequency in the corresponding deposition from the arterial blood.

c. Adventitious Structures.—These formations are in general alike rare in the veins and the arteries (see p. 207), although cancerous disease of the veins presents an exception to this rule.

1. A *fibroid tissue* occurs as an inflammatory product in thickening and sclerosis of the cellular coat of the vein. It is in a great measure the cause of the resemblance to the character of an artery exhibited by the diseased veins.

2. An *anomalous osseous substance,*—the so-called *ossification of the veins.*—It may be distinguished in accordance with its seat into a *peripheral* and a *central* form. It also varies in the character of its substratum.

a. The *peripheral* includes the process of ossification that takes place in an excessive formation of the lining membrane of a vein. It appears, as in ossifications of the arteries, in the form of plates, which, however, for obvious reasons, never attain the thickness of those which occur in the arteries. Its seat is in the venous wall. It is uncommon, and, except in extremely rare cases, very limited in extent. It commonly affects varicose veins, especially the saphena, and we occasionally observe it in the deep veins of the extremities, when they are in contact with ossified arteries, as, for instance, in the internal spermatic vein in males, or in the trunk of the portal vein or its splenic branch (of which we have examples in our museum); they must be ranked amongst the very rarest phenomena.

b. Central ossification includes two forms of concretions, differing in their substrata, namely, phlebolites and the ossification of the coagulum closing an inflamed vein (see Phlebitis) after its temporary metamorphosis into a fibroid string. Both lie in the canal of the vessel, either freely, or coalescing in the modes that have been already described, with the wall of the vein.

3. *Tuberculosis* does not occur either in or on the bloodvessels, as we have expressly remarked in our observations on the endocardium and the arteries.

4. *Cancer* is, on the other hand, by no means a rare phenomenon in the veins. There are two different ways in which it may occur:

a. The walls of a vein are very often perforated by an adjacent cancerous growth, that is to say, the cancer attacks the tissue of the vein, like other tissues, and penetrates into the canal of the vessel. In this way, large veins, as, for instance, the trunk of the ascending vena cava, not unfrequently become entirely closed by exuberant cancerous growths lying on the lumbar portion of the vertebral column; and the same occurs in the veins of a cancerous organ, as, for instance, the renal veins, the portal vein, the hepatic veins, &c.

b. There is another form which is rarer, but of much greater interest, which may be termed *primary cancer*, to distinguish it from the former. It occurs in a vein, generally one of the larger ones, as a broad or narrow, pedicled, roundish, pear-like, single or lobular adventitious mass, or as a cylindrical mass adhering to the whole or greater portion of the circumference, and closing the vein. It sometimes is attached only loosely to the lining membrane by means of a gelatinous blastema, or its parenchyma penetrates into the tissue of the venous wall to various depths. According to circumstances, of which the most important is the duration of the existence of the adventitious product, it presents the appearance either of a fibrinous coagulum, or of an aggregation of primary cells in various stages of development, the whole mass commonly presenting the characters of medullary cancer. This form of cancer never occurs except in association with a large exuberant cancerous growth in some other organ, or when there is a widely diffused and rapid formation of cancerous tumors in many organs, especially of intense medullary cancer (encephaloid). It scarcely ever occurs in the organism, except as a secondary formation. The lymphatics in the vicinity of the original adventitious structure are very often plugged up and closed by the medullary cancer.

This cancer of the veins doubtless occurs through the absorption of cancerous matter by the lymphatics and veins into the mass of the blood; and, independently of the latter, it may be of spontaneous origin, and may be the result and expression of the highest degree of endogenous disease of the fibrin: the circumstances under which this disease has been observed accord with both these modes of origin.

Moreover, we must here include the coagulum which closes the veins and originates from the absorption of cancerous or ichorous matter—the phlebitis arising from cancer.

§ 4. *Anomalies of Calibre.*

A. *Dilatation of the veins, Phlebectasis, or Varicosity in its wider sense*, is, as is well known, an anomaly of great importance in medicine, whether it be general, occurring as a preponderance of the venous system, or whether it be partial and local. It has attained this importance through certain views regarding its etiological relations, derived from clinical observations: and it has maintained this importance, although the more remote cause to which it has been referred,—the venosity,—notwithstanding all endeavors to elucidate it, has been as yet but little understood in reference to local venous dilatations, and to the formation of true varices. With the view of indicating the necessary points regarding the causes of phlebectasis, we shall first treat of its anatomical relations.

The Form of the Phlebectasis especially belongs to the domain of anatomico-pathological inquiry. Two principal forms may be distinguished.

1. The dilatation of the vein may be uniform and cylindrical, the distended vessel running in a straight line; its coats are either attenuated or not of a thickness corresponding to the calibre; or, on the other hand, they may be visibly hypertrophied and thickened.

2. The dilatation may be irregular, attacking the vein merely at certain spots, or at all events very much preponderating at them. It then includes *varicosity*, or *varicose dilatation of the veins*, in the stricter signification of the term.

The venous coats, if not absolutely are relatively thin in proportion to the calibre of the vessel, but after this condition has existed for some time, they very commonly thicken. Varicosity includes two varieties, which merge into one another, and are often simultaneously present in the same vessel.

a. The vein, while it becomes dilated, at the same time becomes elongated, and assumes a winding course, at first forming slightly concave arches; but gradually, as the vein expands on the convex side of the arch, the curve becomes sharper, and the dilatation exhibits itself as a sinuosity *on only one side* of the vein, while the opposite wall of the vessel is usually tense, but sometimes lying in folds. As this process may be several times repeated on the same side, or as it may alternately occur on either side, the course of the vein resembles the convolutions of the intestine, or, even more closely, those of the vesiculæ seminales; indeed, when the vein is sharply bent upon itself, projecting ridges are produced on the interior of the sinuosities—duplicatures of the venous wall, which give to the interior a partitioned structure. We have already (in p. 257) pointed out the derivation of a remarkable degree of dilatation (varicosity) of this kind, both in reference to its winding course and the partitioned nature of its internal structure, from phlebitis, ending in imperfect obliteration.

b. When a strongly marked lateral sinuosity occurs at a circumscribed spot, a true *varix* is formed. It is a saccular expansion, which either lies with a broad base upon the vein, or is sometimes connected with the vessel by a neck or pedicle; in either case communicating with the interior by a wide or narrow opening. In relation to the construction of the walls of the varix, they are formed of all the coats of the vein; or else we observe the circular fibres separating from one another at certain spots, when the inner coat becomes so fused with the cellular coat, that if a further (secondary) bulging occur the varix assumes a *hernial* character. The size of the varix ranges from that of a hempseed to that of a walnut or a hen's egg, or it may be even larger.

The varix is, as a general rule, originally roundish, but by further irregular dilatation at particular spots, it may assume an irregular, externally lobulated form; while internally it presents a cellular, many partitioned structure. Varices of smaller and more delicate veins not unfrequently present blackberry-like tumors.

The *valves* exhibit a different relation in phlebectasis. They at first offer such opposition to distension, that they limit and bind down the varix; it is not, however, by any means invariably at the valves that we find these constrictions occurring on varicose veins. The valves increase in size, to a certain degree, with the dilatation of the vein, but after a time they cease to increase, and are no longer capable of closing the enlarged vessel; they then lie, in a state of tension, transversely across the tube of the vein; or they are drawn in an eccentric direction (towards the periphery); or, finally, they may be torn, in which case they float loosely in the vessel, or they may be almost destroyed, so that we can detect mere traces of them.

If repeated inflammatory attacks have not fixed the varicose veins in their bed of cellular tissue, and made them coalesce with the adjacent structures, they may be often readily detached, and easily raised from it, leaving furrows and cavities, with smooth even walls.

The following are the *sequelæ and results* of dilatation of the veins, especially of varicosity.

In varicose veins, coagula of blood are occasionally observed in the form of roundish, oval, fusiform, detached plugs, or of cylinders which close the vessel. These are generally again dissolved after a shorter or longer period, and taken up into the mass of the blood; but, as new impediments, they sustain and increase the varicosity. These coagula appear to be very frequently produced in the varicose plexuses of veins, which often occur in the pelvic viscera, new clots being in the act of formation, while the older ones are undergoing solution. They lead, in the manner which has been already described, to the formation of phlebitis.

In large varices, especially such as are connected by a pedicle, we sometimes find stratified coagula of fibrin, as in aneurisms. Such varices are sometimes shut off from the calibre of the vein by a newly formed inner venous coat, investing the last formed stratum of fibrin; or they become separated from the vessel by a prolongation of their pedicle, and a closure of its calibre. In the latter case they degenerate into fibroid capsules.

Varicose veins often, however, open, either externally or into mucous canals and cavities, after they have become imbedded in the tissue of the general investments, or of a mucous membrane which has become extremely attenuated and distended over them; the vein, and the superjacent tissue with which it has coalesced, and which has simultaneously been affected with inflammation, undergoing laceration together.

The varicosity induces stases, which are either transient or persistent, according to circumstances, and are occasionally marked by exacerbations, in the capillary system; and these are followed by various forms of œdema, hypertrophy, increased secretion from the mucous membranes (blennorrhœa), and inflammation, especially of the cellular tissue and the skin, terminating in hypertrophy and sclerosis of the cellular substance, as well as in ulceration.

Moreover, varicose veins are very frequently subject to inflammation. This tendency is most probably based on the stasis which is induced by the varicosity in the vasa vasorum. In accordance with this view, it is generally a chronic affection, whose product is for the most part confined to the cellular coat of the vein and the adjacent cellular tissue, in which it causes hypertrophy and condensation, glues the vein to the neighboring textures, and renders its walls rigid.

The cases, however, in which the inflammation is acute, are sufficiently common. It then deposits a variety of products on the inner surface of the vessel, and its results are either atrophy and obliteration of the varicosity, or acute suppuration.

Varices, moreover, sometimes begin to ulcerate from their cellular coat, and from the contiguous cellular tissue; the ulcerous perforation of the varix from without commonly impinges on a coagulum of blood, and hence the symptoms of pyæmia do not ensue.

The *distribution* of phlebectasis is extremely various, and is determined by different causes. It may uniformly attack the whole venous system; or, on the other hand, it may be limited to almost any section of it in the form of varicosity. Varicosity is either originally developed on the small veins—the venous radicles,—and extends from thence towards the trunks; or it may originate in the latter, and retrograde from thence to the ramifications.

The *causes* of phlebectasis are usually sufficiently obvious; as, for instance, mechanical impediments to the circulation of the blood in consequence of contractions in the openings of the heart; or pressure on a venous trunk; pressure on a large vein by a tumor, which may close or even obliterate it; a flow of arterial blood into a vein; a position of the body interfering with the proper reflux of the blood into certain parts, in which the course of the veins lies in an opposite direction to the force of gravitation; excessive activity of an organ, accompanied with its enlargement and hypertrophy; adventitious products, in which vessels of considerable size have been produced—that is to say, the insertion of a new vascular apparatus into the original venous system of a part; repeated hyperæmia and inflammation of an organ; or inflammation of the vein itself. The impediments of a mechanical nature have always been regarded as of the greatest importance.

Cases are, however, occasionally met with, in which none of the abovementioned causes can be detected, and, indeed, where no mechanical hinderance can be perceived. Such cases have recently been often brought forward with the view of opposing the theory of a mechanical impediment, and of establishing other theories of varicosity. It is necessary that we should mention the grounds alleged in relation to this point.

1. The cases in which no mechanical impediment exists are by no means rare.

2. If the veins of dependent parts are more frequently varicose than others, this does not explain why they are oftener varicose in one limb than in another; or why this condition especially occurs in a particular part of a limb, and why neighboring veins, under the same conditions, are not similarly affected.

3. Moreover, varices also occur, without any mechanical impediment, in veins in which the direction of the current coincides with that of the force of gravitation, as, for instance, in the veins of the head and neck.

4. In women, varices often occur in the legs during the early stages of pregnancy, before the uterus can exert any appreciable impediment to the circulation.

5. When constipation induces hemorrhoids, they may be as much ascribed to the irritation and congestion which it excites as to the impediment produced to the course of the blood, for otherwise the hemorrhoids would disappear when the bowels were freely moved, which is not the case.

6. When veins running towards a tumor become varicose, it cannot always be shown that they all undergo compression by the tumor; moreover, the blood might make its way by numerous anastomoses with deeper and non-dilated veins.

7. The blood, far from stagnating, rushes through the varices with great energy, which thus increases the difficulty of checking hemorrhage from them.

8. The blood contained in varices is brighter than venous blood, resembling a mixture of arterial and venous blood.

9. When arterial blood makes its way into a vein, the latter becomes varicose.

10. Varices sometimes present pulsations isochronous with the heart-stroke.

11. When a main artery is tied, in cases of aneurism, we very often see varices disappear in the neighborhood of the aneurism. (Dupuytren.)

12. Most varices extend from the venous radicles towards the trunks, which can only happen in consequence of the extraordinary impulse that the blood in those vessels receives from the heart, or of the blood flowing from the trunks towards the branches, which is always the case where a morbid formation acts as a centre of attraction for the blood.

Briquet attempted, in an unsatisfactory manner, to explain the formation of varices from an excessive fulness of the subcutaneous veins of the lower extremities, arising from the contraction of the muscles, which drive back the blood from the deep to the superficial veins; and Rima subsequently maintained that varices of the lower extremities were dependent on a retrograde motion of the blood, so that the blood flows back from the femoral into the saphena vein, and is driven from the inguinal region towards the feet by a force which is peculiar to this vein. Pigeaux, from a more general consideration of the same facts, believed that he had solved the difficulty by the assumption that varices anastomose with arteries.

If objections may be raised against some of the above-mentioned facts in opposition to the theory of a mechanical impediment, we are nevertheless convinced of the untenability of the latter view. We have, therefore, after much experience, adduced other causes in explanation of dilatation of the veins. There are, however, always cases occurring in which these cannot be detected; and as varicose veins present many symptoms which are hitherto perfectly unexplained, the theory of phlebectasis is still deficient in an important part. Now, although Pigeaux's view is not yet actually proved to be correct, and is only based on the theoretical application of certain phenomena of varix, it is so far worthy of consideration, as it removes the whole question into the department of anatomy, and thus affords facilities for renewed and more profound investigations. Thus only is it possible to obtain a well-grounded view regarding the correctness of a theory which has become popular in Germany, respecting the production of a congenital (hereditary) or acquired disposition to general or local phlebectasis, respecting the production of a persistent or periodically recurring blood-crasis and its localization, the production of peculiar critical events in the organism, in consequence of many acute processes of plethora and stasis (venous congestion) occurring from time to time in certain portions of the venous system, regarding the high importance of local phlebectases arising from them, &c.—a theory which many, even from the want (as yet) of any material information on the subject, are inclined to regard as a deception and a fiction.

Phlebectasis is most common in the prime of life. Some forms, however, develope themselves earlier, and all may persist to extreme old age. Many forms of phlebectasis may attack both sexes; some, however, chiefly, or even exclusively, affect only one sex.

Of the local phlebectases some require a special notice, in consequence of the frequency of their occurrence, and others from the annoyances and pains to which they give rise. Several very often occur in one and the same individual: whether the frequently isolated occurrence of a definite species of varicosity—as, for instance, of varicocele (Landouzy), and whether the high development of one species, in association with a low degree of another, is indicative of a vicarious action, and must be referred to a deeper cause, are points which must remain for the present undecided. As a general rule, phlebectases are almost entirely confined to the lower half of the body, and their occurrence in the upper half, must be regarded as altogether exceptional. It is almost invariably the superficial or cutaneous veins which become varicose in the lower extremities.

a. Varicosity of the veins of the lower extremities is very frequent, and may be observed in all possible stages. It attacks the system of the saphenous veins, and especially the trunk and branches of the internal saphenous vein. According to our observations, it attacks both sexes with equal frequency. A protracted, uneasy, and upright position of the body is undoubtedly a very frequent cause of this affection, although the cases are not uncommon in which it cannot be traced to this origin. In women, especially amongst the working-classes, it is often ascribed to frequent pregnancies. Hasse remarks that, as a general rule, the dilatation commences in men in the trunk or principal branches of the internal saphena vein, while in women it usually begins in the finest cutaneous branches on the inner side of the limb. The disease is commonly first developed on the lower part of the leg, and from hence it extends to the thigh, where it is for the most part limited to the trunk of the saphena. We believe, however, that, especially in women, the cases are not very rare in which the varicosity first appears and predominates in the femoral region. It often attacks both limbs, but more commonly only one. The dilated venous reticulations either lie loosely in the panniculus, or they are imbedded in a callous, thickened, infiltrated cellular substance, with which they coalesce; while the venous coats themselves are thickened, and have a rigid appearance when cut through. This condition of the saphena vein may give occasion to the entrance of air into it, if it be opened, as is shown by an operation of Dupuytren's. Coagula of blood are very commonly formed in these veins, although it is extremely seldom that we find phlebolites in them.

Varicosity is followed by œdema, hypertrophy, repeated inflammations of the cellular tissue terminating in indurations, and inflammations of the skin, which cause it to coalesce with the subjacent cellular tissue, and give rise to an excessive formation of epidermis, and ulcerous fusion of the tissue—the so-called varicose ulcer, which is distinguished by the callosity and livor of its edges, the production of a purulo-serous corroding secretion, lax and bloody granulations, its sinuous and sometimes serrated form, and, finally, by its obstinacy. The varicose veins at its base, or in its borders, are sometimes corroded, and give rise to exhausting, or even fatal hemorrhage.

When the pressure of the varices destroys the fascia under which they lie, they become deposited in the subcutaneous cellular and fatty tissue, and finally in the true skin itself, which now becomes extremely attenuated, inflames, and gives way, causing a hemorrhage which also may prove fatal.

We very frequently meet with inflammations of these varicosities, which sometimes terminate in resolution, sometimes in obliterations and atrophy—a termination which it is the object of the various operations for varices to induce,—and sometimes in purulent exudation, which may occasionally prove fatal, by giving rise to pyæmia.

b. Varicocele (Circocele), varicosity of the veins of the spermatic cord and of the testicle is usually developed during the period of puberty, and is commonly regarded as of more frequent occurrence on the left than on the right side—a view which is borne out by our own somewhat limited experience. It begins with a uniform, cylindrical dilatation of the veins in the spermatic cord; these gradually assume a character of true varicosity, which extends downwards towards the testicle, and often affects the scrotal veins (*circocele*, according to Velpeau). It is frequently combined with consecutive hydrocele, and in its higher degrees is followed by atrophy of the testicle, and at the same time by mental disease.

The varicose veins are here rarely attacked by inflammation; and phlebolites are seldom produced in them, although not so seldom as in varicosities of the saphena.

c. Varicosity of the veins of the rectum, constituting *Hemorrhoids*, is, next to the preceding, undoubtedly the most frequent form, and is that which has been commonly regarded as the expression or crisis of a special (often hereditary) diathesis. Although we are now fully aware that too great a latitude has been allowed to the theory of hemorrhoids, yet hitherto no positive facts have been brought forward on which a better theory can be founded. It is worthy of remark, that even to the most recent times, erroneous views have often been promulgated regarding the nature of local hemorrhoidal disease, in consequence of our departing from the original view, that hemorrhoidal tumors are dilated varicose veins. We have convinced ourselves on innumerable occasions, that hemorrhoids are ordinary varicosities; and if we must, on the one hand, reject the opinion that they are produced by extravasation and sanguineous infiltration of the cellular tissue (Recamier, Gendrin), we, on the other hand, find no reasons for ascribing to them a peculiar erectile nature (Cruveilhier).

This varicosity affects the small veins, which freely anastomose with one another at the extremity of the rectum, and which lie thickly imbedded under the mucous membrane in the subjacent cellular tissue encircling its lower expanded portion. A certain number of these become dilated and stand out, sometimes as separate knots, sometimes as a row of nodular swellings, thus causing the lower border of the rectum to protrude; or they may be within the rectum: it is only rarely, however, that they occur above the external sphincter. In a less developed stage of the disease, and at the beginning, they are small; but, after repeated swellings, they attain the size of a bean, a hazel-nut, or a walnut. They are then protruded from the gut when the bowels are moved, the rectum is choked up with them, and they are constricted by the border of the anus, which not unfrequently gives them a pedicled form. At first the swelling disappears without leaving a trace; the oftener, however, that they swell, so much the more is the mucous membrane of the rectum, in which they lie, left in persistent folds and elongations, which project externally; and finally, when repeated inflammations have taken place, a recession of the knots (varices) no longer occurs, and they assume a condition which renders them capable of still further enlargement, but not of any diminution.

Hemorrhoidal knots at first form roundish, simple sacs, which, however, afterwards become sinuous, and partitioned in their interior; their walls at first are thin, and collapse on being cut; they gradually, however, become thick and rigid, in consequence of repeated inflammations. One, or very commonly several fine venous branches open into the cavity of each knot, whose lining membrane is undoubtedly composed of the inner coat of the veins. The veins adhere, as a general rule, very tenaciously to the mucous membrane of the rectum, but they may be at first easily detached from the cellular tissue and the muscular coat. The cellular tissue lying between them is, like the mucous membrane, in a state of injection, reddening, and tumefaction—in short, in a condition of stasis.

The hemorrhages which occasionally proceed from the varicose swelling of the veins in the rectum, are doubtless dependent in some cases on the rupture of a varix, and of the tense and thin mucous membrane covering it. True capillary hemorrhages of the mucous membrane itself are, however, incomparably more frequent.

Hemorrhoidal varices usually contain coagula of considerable size, which dissolve, without, however, often giving rise to the formation of phlebolites.

The stasis to which we have referred is gradually followed by a thickening and condensation of the surrounding cellular tissue, a condition which is induced in a still higher degree by repeated inflammatory attacks. It often gives rise to inflammation, whose products induce a thickening and sclerosis of the coats of the veins and of the surrounding cellular tissue, and cause their fusion with each other. This inflammation sometimes also simultaneously deposits its products within the vessel, in which case it leads to obliteration and atrophy of the varix. In rare cases, especially after the application of a ligature to the hemorrhoidal knots, there is a purulent exudation, which extends to the larger veins, and may induce fatal pyæmia. More frequently, however, the inflammation of the surrounding cellular tissue gives rise to suppuration, abscess, destruction of the varix, ulcerous perforation of the mucous membrane, and finally to fistula in ano.

Amongst the consequences of hemorrhoidal disease, we must also mention habitual hyperæmia of the mucous membrane of the rectum, with swelling and blennorrhœa, prolapsus ani, hypertrophy and stricture of the sphincters, sclerosis of the cellular tissue around the rectum with paralysis of the sphincters, and hemorrhoidal ulceration of the rectum.

These ulcers at the extremity of the rectum are analogous to the varicose ulcers which occur on the feet, and often bleed very freely from corrosion of varicose veins, or even of small arteries.

Hemorrhoids sometimes depend on certain obvious impediments in the portal system, in the ascending vena cava, or in the heart; but in many cases no such hinderance can be detected.

We are as unable to give a strict scientific explanation of the connection between hemorrhoidal disease generally, suppressed hemorrhoids, &c., and the various so-called hemorrhoidal anomalies—as, for instance, congestions, hemorrhages, impetigo, catarrh, gout, lithiasis, &c., as we are to comprehend the true nature of what is called the hemorrhoidal diathesis.

d. Varicosity of the vesical veins (known also as *hemorrhoids of the bladder*) in the male sex affects the veins of the prostatic and vesical plexuses; the branches of the latter on the neck of the bladder and around the vesiculæ seminales, external to the muscular coat, being, in general, especially dilated. In the female sex, the veins of the vagina are thus affected in addition to the vesical plexus; and the varicosity extends over the veins of the bladder to the *veins lying between the broad ligaments of the uterus*. This form of disease attains a very high degree of development, and is common in advanced life and old age. Phlebolites are nowhere so frequent, so numerous, or so large, as in the veins we have just mentioned.

The veins situated immediately beneath the mucous membrane of the bladder are less frequently varicose, although we have repeatedly observed this condition, as well as the occurrence of laceration of a submucous varix on the posterior wall of the bladder, with hemorrhage into its cavity. The veins of the neck of the bladder present the only exception; they are frequently dilated and swollen.

Varicosity of the veins of the bladder is often combined with hemorrhoids; in many cases, however, we find nothing but their residue, or they are even wholly absent. We are as yet unable to determine from anatomical investigations whether, in such cases, the varicosity of the veins of the bladder has actually taken the place of the hemorrhoids, and, having thus assumed the same significance, may be regarded as of a hemorrhoidal nature.

e. Although *varicosity of the upper extremities, and of the head and neck*, is less frequent than the above species, we yet occasionally observe this condition very highly developed in the *lips.* It is moreover of rare occurrence in the intestines, mesentery, stomach, and œsophagus. It must, however, be observed that this remark applies only to true varicosity, since a uniform dilatation of the veins is frequently found to exist in a very highly developed condition in these structures in various affections, such as heart-diseases, impermeability of the liver, displacement of the intestines, dilatation and hypertrophy of the stomach, &c. We are, however, able, from personal experience, to confirm the observations of others, who have noticed true varices on the œsophagus, the stomach, and intestines, and even beneath the mucous membrane, which have terminated in laceration and hemorrhage.

f. Varicosity of the Veins of the Pia Mater is an important condition. It has been frequently noticed in the case of drunkards, and more especially after repeated attacks of delirium tremens, and is often combined with cerebral atrophy. The venous trunks of the infiltrated swollen pia mater on the convexity of the hemispheres which open into the superior longitudinal sinus, appear to be simply dilated below the turbid and thickened arachnoid; in the direction of their ramifications, the vessels present, however, a very peculiar appearance, being elongated, and more or less twisted into intestine-like circumvolutions, or even spirally-twisted coils. Similar dilatations not unfrequently affect the veins of the *choroid plexus.*

g. It still remains for us to notice another form of varicosity of the trunk, which affects the *subcutaneous abdominal veins*, and originates in a congenital anomaly of the vascular system. Thus, for instance, there exists in many persons an anastomosis of the umbilical vein, with the veins of the abdominal integument, one of which opens into the umbilical vein. This anastomosis occasions the continued patency of the umbilical vein after birth, and thus maintains on unusual communication between the systemic veins and those of the portal system. In such cases, the veins of the abdomen become by degrees excessively varicose, undoubtedly in consequence of the circulation in the portal trunk obstructing the discharge of their blood through the umbilical vein, or perhaps, even from the entrance of portal blood into the umbilical vein. This varicosity presents the appearance of a wreath-like network surrounding the navel (Caput Medusæ), or of pyramidal tumors at the side of the navel; or, lastly, the veins of the abdomen generally may be varicose in the direction of the loins and the buttocks, as well as towards the lower extremities.

Cases of this kind have been noticed by the older observers, and in modern times by Cruveilhier, Manec, Peygot, and others. We ourselves have noticed the same condition in two men and one woman. In two of these cases the varicosity presented the Caput Medusæ form, and in the third (that of a man) it was present on only one side of the abdomen, from whence it extended down the loins, nates and leg to the foot. The dilated veins contained an innumerable quantity of phlebolites, varying in size from a barley-corn to a bean, which were so densely crowded together as to make the vein feel like a skin filled with shot (see p. 266). It appears worthy of notice that in all the cases of the kind which we have seen, the liver was in an abnormal condition, presenting in one instance granulations, and in two others, a lobulated condition, in consequence of the obliteration of several of the portal branches—adhesive phlebitis of the portal vein.

B. *Occlusion, Contraction, and Obliteration of the Veins.*—To this class belong several anomalies to which we have already referred in the preceding pages; and of which we now proceed to consider the most important.

1. *Contraction and final obliteration of the vein in consequence of persistent compression.*—Such a form of compression is exerted by all tumors generally, but more especially by aneurisms; and we have frequently observed both a threatened and a complete destruction of the calibre of the vena cava superior from aneurisms of the ascending aorta. The vessel is first flattened at the spot exposed to the pressure, and when the latter is increased, the walls of the vein are at length brought into permanent contact with each other, and obliteration is thus established, consisting in a *fusion or coalescence of the lining membrane of the vessel.* The occlusion of the vessel above and below the coalescence by means of a coagulum (thrombus) is merely a secondary and unimportant occurrence, which, moreover, follows the known laws of a thrombus-formation.

2. *Occlusion of the vein from coagulation of the blood.*—To this class belong, independently of the coagula occasioned in varicose veins by the retarded flow or stagnation of the blood, the obstructing coagulum in phlebitis, and the coagulation arising at any part of the venous system from a diseased condition of the blood. Both (see pp. 257 and 260), may induce more or less complete *obliteration* of the vein. This is the most frequent form of occlusion and obliteration of the veins.

3. *Occlusion arising from phlebolites* seldom induces entire impermeability.

4. *Occlusion of the veins from cancerous secondary formations* is not of very rare occurrence, even in the venous trunks. (See p. 267.)

The ordinary results of these anomalies are *œdema and dropsies;* the latter are, however, retarded in their development and healed by the establishment of a collateral circulation (even where the main trunks are closed) through the anastomoses of numerous veins. The obstruction which the valves must here present (Stannius) is undoubtedly overcome in many cases, or so far neutralized, that a collateral circulation is established, as in the arteries, by the anastomoses of very minute vessels having no valves. The closure of the portal vein can scarcely be compensated for, notwithstanding the numerous anastomoses of its roots with the systemic veins, and hence the dropsy which it induces does not admit of cure. Closure of the vena cava inferior is compensated for by dilatation of the azygos and hemiazygos, and anastomoses of the epigastric vein with the internal mammary, and of the subcutaneous abdominal veins with the axillary: it is on the other hand less easy to compensate for the closure of the vena cava descendens, especially when the mouth of the vena azygos has at the same time become impervious; the collateral circulation is carried on by means of the venous plexus of the spinal canal and its anastomoses with the subclavian and hypogastric veins, by means of the anastomoses of the phrenic veins with the vena cava ascendens (and even with the great coronary vein of the heart, as occurred in a case of Reynaud's), and lastly, by means of anastomoses of the axillary and internal mammary, with the epigastric and circumflex iliac veins.

In rare cases we have also seen the circulation re-established in closed veins by means of canals, which have become developed near the centre of the occluding plug. This phenomenon either depends on the channelling of the thrombus—a process which we have already described in p. 248—or it is due to the disintegration of the central portion of the coagulum into a molecular mass, which gradually becomes taken up into the blood, while the outer layers assume a fibroid character and coalesce with the venous walls. We have never observed such cases, but they have been described by Carswell and Hasse.

§ 5. *Separation of Continuity.*—Under this head we include *the various wounds and spontaneous lacerations* of veins. The *former*, as is well known, heal by adhesion of the edges in cases of small incised or penetrating wounds; in cases, on the other hand, where the wound is larger, or where the vein is completely cut through, the cure is effected by obliteration of the vessel, consequent on a process which is generally much the same as that which occurs after an artery has been cut through.

Spontaneous lacerations of veins, if we except the bursting of true varices, are rare, in comparison with the lacerations of arteries. We have, however, observed them in the trunks of both venæ cavæ, in the azygos, the pulmonary, and other large veins.

SUPPLEMENT.

Anomalies of the Small Vessels and Capillaries.—Although we deem it necessary to refer specially to the anomalies of these vessels, we must observe that we are entering upon a field of inquiry which has hitherto only been partially cultivated in reference to the general anatomy of certain processes. In the following remarks, we purpose referring to what has been already stated, both generally and specially, regarding the diseases of the vessels.

We therefore omit to notice the formation of new vessels, which occurs under certain conditions, as well as the atrophy of the capillaries, which presents itself under other conditions, and proceed at once to the consideration of the following anomalies.

§ 1. *Anomalies of Calibre and especially Dilatation.*—Under this head there are commonly placed *Aneurism by Anastomosis*, *Teleangiectasis*, *Fungus hæmatodes*.

Aneurism by Anastomosis consists, according to J. Bell, in dilatation of the small arteries and their anastomoses, giving rise to a pulsating, more or less defined, tumor; it is separated from the venous system by normal capillaries; the dilatation sometimes, however, extends to these vessels, and, finally, to the veins.

Teleangiectasis, on the other hand, consists in a dilatation of the capillaries, and appears sometimes in the form of slightly elevated patches, which may be smooth or may present inconsiderable nodules, and sometimes in the form of roundish, nodular, and lobulated smooth tumors, which are either of a bright-red color, or of a darker, purple or cherry-red tint, and are either tough and elastic, or soft, and of a doughy consistence. They are capable of swelling and of undergoing a considerable collapse; and hence the name of *erectile tumors;* which Dupuytren has applied to them. We must, however, remark, that there is no analogy between the arrangement of their vessels and that which occurs in the erectile organs. Teleangiectasis may certainly in some cases be developed chiefly on the arterial side of the capillaries, and in other cases on the venous side; and its form may in part be referred to its position, for we regard those that extend superficially as chiefly of an arterial nature, and those which become developed into tumors as especially of a venous nature. It occurs most commonly in the tissue of the general integuments, and in the subcutaneous cellular substance; and further, besides those cases in which it is continued from these to the neighboring mucous membranes, it is also developed independently upon the mucous membranes, as, for instance, those of the intestinal canal; and, lastly, in rare cases in other tissues, as in the muscular substance, &c.

It is in general congenital, although it is capable of further development after birth. In some rare cases it even originates in later periods of life.

The structure of congenital teleangiectasis is mainly to be referred to the transition of the dilated extremities of arteries into saccularly formed dilated venous radicles, from whence a varicose condition extends for a considerable space to the larger ramifications. The convolution of dilated vessels is held together by means of a loose and partially embryonic cellular substance. The above remarks give the main points of the opinions hitherto maintained in reference to dilatation of the minute vessels and the true capillaries—more especially when the disease is of a congenital character. But these views undoubtedly require much consideration. A similar observation applies equally or even more forcibly to the teleangiectases, which are observed to arise in different and more advanced periods of life, as a consequence of repeated inflammations, and of loosening and rarefaction of the tissue in the neighborhood of ulcers, and in and around the most various new formations, &c. The dilatation of the existing capillaries, which has commonly been considered as a sufficient cause for this anomaly, is certainly, in many cases, inadequate to explain its existence, and we find that, in addition to this condition, there occurs simultaneously a new formation of vessels,—a subject which we have already considered generally in its relation to the original vascular apparatus.

Fungus hœmatodes, which does not consist in a mere dilatation of the vessels, has been fully noticed as a secondary formation in the General Anatomy.

The present notice would be incomplete, were we to omit all reference to a morbid structure, which has been generally regarded as belonging to the teleangiectases, and from which it has been supposed, on somewhat obscure grounds, to be developed. It does not appear to us to belong to these affections, and we are rather disposed to regard it for the present, at all events, as a new formation. It includes *cavernous tumors*, having a cellular structure similar to that of the corpora cavernosa. They are composed of a cellulo-fibrous tissue and a lining membrane investing the interstices, and enclosed in a dense cellular capsule. These structures admit of being separated from the organs in which they are seated, are capable of tumefaction, and may be injected by a vein. They occur in the form of the so-called secondary spleens (which, according to Andral, are of a placenta-like texture) in the subcutaneous cellular tissue, and are of especially frequent occurrence in the liver; we have also observed them in the cranial bones, and in the pia mater. They sometimes contain sand-like concretions, which may be regarded as similar to phlebolites.

§ 2. *Separations of Continuity.*—These are very numerous in the capillaries, or arise from cuts or from lacerations induced by contusions and shocks. They result in an extravasation of blood into the tissue, and hemorrhage externally or into different cavities and canals.

A higher degree of interest must be attached in a pathological point of view,—1, to spontaneous laceration of the capillaries in apoplexies (parenchymatous hemorrhages) of the different organs and tissues, arising from hyperæmia (vascular apoplexy), which, although formed in the usual manner, has been developed beyond a certain limit;—and 2, to spontaneous lacerations resulting from a morbid brittleness of the walls of the vessel, or from a similar condition of bogginess in the tissue affected by the hemorrhage, as, for instance, the substance of the uterus.

§ 3. *Anomalies of Texture.*—We have already considered the relation of the capillaries in hyperæmia, stasis, and exudation (inflammation), as well as in their modes of termination, in induration (atrophy of the tissue), in suppuration, in gangrene, and in other conditions. Although, as we must again, specially remark, these vessels cannot themselves be inflamed, they yet become the seat of an inflammatory process, and a centre for the development of its products, the deleterious effects of which they are the first, under the appropriate conditions, to experience.

The small veins and arteries beyond the capillaries are affected by the condition of the surrounding tissue, even where they had not themselves participated in the process of exudation: for their delicate and permeable coats are saturated and permeated by the product of the inflammatory process, on which, at least in part, the ordinary coagulation of blood within them and their occlusion depend. There then follows either a solution of the occluding coagulum and a liberation of the calibre of the vessel, when resolution of the inflammation occurs in a comparatively large vessel, or persistent obliteration and atrophy or a suppurative destruction of the vessel, when the inflammation terminates in induration or suppuration.

In connection with the subjects discussed under this head, we must especially refer to *capillary phlebitis*, although we must confine ourselves to little more than a recapitulation of what has been already stated.

Capillary Phlebitis, if it were a true inflammation, should rather be called *Capillary Angioitis;* but inasmuch as it does not in reality consist in an inflammation of the capillaries, neither of these names is applicable to the affection. It essentially and originally consists in no textural change of the capillaries, although it very often gives rise to their atrophy or ulcerous destruction. We have already frequently referred to it, both in the general anatomy, and in different chapters of the special anatomy. It essentially consists in a coagulation of the blood in some portion of the capillary system, dependent on a spontaneous disease of the blood, or on its infection from some cause, and is analogous to the phlebitis induced by coagulation. This coagulum, which closes these vessels, at first appears as a dark-red infarctus in the affected parenchyma, and subsequently becomes decolorized, and undergoes various metamorphoses. If it be not gradually dissolved and absorbed in a finely comminuted state into the blood, it *either* shrivels, and assumes a rusty-brown, yeast-yellow, or perfectly bleached appearance, and the whole terminates in persistent obliteration of the affected capillaries (the adhesive process), associated with atrophy of the parenchyma, which is converted into a white fibroid (cellulo-fibrous) callus; *or* it undergoes, in general with considerable rapidity, a purulent and usually acute ichorous, gangreno-ichorous septic fusion, associated with a yellow, or dirty green, or brown discoloration. The walls of the vessels and the parenchyma participate in the same process, which finally results in the formation of a purulent or ichorous abscess.

Sometimes the coagulum, after it has become yellow, undergoes partial or entire cretefaction.

An exudation into the parenchyma doubtless occurs simultaneously with this process. As compared with the same process in a larger vessel, we regard it as an unessential occurrence, merely depending on the permeability of the walls of the vessels.

To these processes, considered with reference to the general condition, or to a focus of infection giving rise to it, we commonly apply the term *metastases;* or in the lungs, we term them *lobular* processes, in consequence of their usually inconsiderable extent and well-marked limitation.

Around their margin we commonly find a true secondary (reactive) inflammation of the tissue, which the experienced observer may tolerably readily distinguish from the original centre of inflammation, especially in the parenchymatous structures, which—as, for instance, in the case of the lungs—undergo a peculiarly striking degeneration (hepatization). Immediately around the inflammatory centre we not uncommonly meet with capillary hemorrhages, and suffusions of the tissue.

They occur in all tissues, but are especially frequent in certain very vascular organs which take part in hæmatosis, as the lungs, the spleen, the kidneys, and the liver. They are principally distinguished by their generally considerable number, by the roundish or (in the case of the spleen and kidneys) the wedge-like and angular form of their central part, and from their being seated on the periphery of the above-named organs.

When the affection terminates in obliteration or atrophy, pit-like, contracted depressions are formed on the organ, which are generally the more numerous the nearer the morbid changes are to the surface.

Cruveilhier also treats of a *hemorrhagic capillary phlebitis*. In reference to this name, we will only remark that the capillaries which are the seat of what is termed capillary phlebitis, cannot give rise to hemorrhage. In the lower extremities of aged persons suffering from dyscrasia, we meet with coagula, especially in the subcutaneous veins, which extend from the trunks into the branches, and may possibly at some spots affect the capillaries. The cellular tissue is then the seat of a spontaneously distributed suffusion; it appears sometimes to contain an extravasation of loosely coagulated blood, without any apparent degeneration of texture or cohesion; and sometimes it and its contained blood represent an apparently firm, but easily torn, friable, dark-red clot. We believe that this process consists in a spontaneous coagulation occurring within, and closing the trunk of the vein and its branches, and giving rise to diffuse capillary hemorrhages, in consequence of the impediment which is presented by the occlusion to the emptying of the capillary vessels, and is proportional to the extent of the coagulation, a phenomenon which we observe, on a small scale, in almost every phlebitis—that is to say, in the form of small ramifying hemorrhagic centres along the occluded vein, and, as we have already remarked, around the seat of capillary phlebitis.

Excessive Deposition of the Lining Membrane.—It is an unquestionable fact, that very minute arteries undergo this form of disease, since their coats lose their transparency, and become opaque and thickened either uniformly or at particular parts; and, instead of remaining soft and flexible, become rigid and brittle, and not unfrequently ossified. We feel convinced that a similar condition exists in the true capillaries; and it is moreover probable, that the anomaly is here less in degree, since the arterial portion of the blood is expended in the process of nutrition.

This condition, which is duly considered in the Diseases of the Arteries, is very important, since, in consequence of the thickening of the walls of the vessels, it impedes their permeability and the nutrition of the tissues; since it also induces partial occlusion, or even perfect obliteration, of the vessels; and since, finally, it predisposes the vessels to laceration. Amongst its results are atrophy of the organs, spontaneous gangrene (mummification), and hemorrhages.

It is especially observed in the brain, in association with ossification of the trunks of the vessels at the basis cerebri, and in the uterus. On making horizontal sections through the hemispheres of the brain, and passing the finger over the cut surface, we sometimes feel roughnesses, corresponding to the exuding blood-spots, and caused by the ossification of very minute arteries.

Adventitious Products.—We have already explained the relation in which the minute and true capillary vessels stand to the different adventitious products, and the changes which these vessels undergo.

V.—ABNORMAL CONDITIONS OF THE LYMPHATIC SYSTEM.

A. *The Lymphatic Vessels.*—We shall pass over the anomalies which the great lymphatic vessel, the thoracic duct, presents at its origin, in its course, and at its mouth, and proceed at once to—

§ 1. *Anomalies of Texture.*

a. Inflammation.—Our anatomical knowledge of inflammation of the lymphatic vessels (*lymphangioitis*) is very deficient in several points, which is in part owing to the delicacy and inaccessibility of the lymphatic vessels, and in part to our imperfect knowledge in relation to their function, and to the extent to which they can absorb heterogeneous substances. In reference to the fine ramifications of the lymphatics, they are so far positively affected in every inflammation of the tissue, that at their numerous points of contact with the products of the inflammatory process, they absorb morbid matters by imbibition into their cavities, and, according to circumstances, sometimes lose their permeability by the swelling of their coats or the coagulation of their contents, and sometimes present torn walls. It is on the absorption of these morbid matters that the consensual inflammatory swellings of the lymphatic glands pertaining to the inflamed organ are based. In inflammations with copious exudation into the tissue, the lymphatics undoubtedly sometimes undergo a transitory occlusion, and sometimes a persistent atrophy where the inflammation terminates in induration; when there is purulent, ichorous fusion of the tissue, they suffer a destruction corresponding to the extent of the process.

It is more easy to observe the manner in which the larger lymphatic vessels are affected by inflammation, but even here there are difficulties to which we will refer after we have considered inflammation of the lymphatic vessels in a purely anatomical point of view.

Lymphangioitis presents the following persistent signs:

a. Injection and *Reddening* of the cellular coat of the vessel; we very often find *small ecchymoses* on it, and on the inner coat. In other respects the vascularization is very frequently insignificant, and sometimes none can be detected.

b. Infiltration of the cellular sheath with serous, sero-fibrinous, purulent moisture, and swelling.

The simultaneous vascularization and infiltration of the surrounding cellular tissue are often very strongly marked; abscesses often occur at various points along the vessel.

c. The inner coat is lustreless, dull, villous, easily wrinkled, and at some parts presents a red or bluish-red speckled appearance from the ecchymoses seen through it.

d. The wall of the vessel is consequently thickened, while its coats become *loosened* in texture, easily lacerable, and removable in layers.

e. The vessel is *dilated* and *varicose*.

f. Exudation occurs in the form of more or less opacity, of distinct coagulated flocculi, or even of larger occluding coagula, or of pure pus, in the canal of the lymphatic vessel.

Whether the occluding coagula, like those in inflamed bloodvessels, notwithstanding the slighter coagulability of the fibrin in the lymphatics, and the lesser exudative tendency of the lymph, may not sometimes be coagula derived from the lymph and not solidified exudations, cannot at present be decided.

Inflammation of the lymphatic vessels may terminate in *Obliteration* or *Suppuration*, as well as in *Resolution*.

1. The lymphatic vessel may close around a coagulum adhering to the inner coat of the vessel and metamorphosed into a fibroid string. We have observed the thoracic duct in a phthisical patient, who was worn to a mere skeleton, present an obliteration of this nature and a conversion into a solid cord.

2. The lymphatic vessel may suppurate, and this ensues not so much from the interior and from the purulent exudation deposited in its canal and in its coats, as from a neighboring abscess denuding and destroying the vessel. The lymphatic vessel then lies as it were in the walls of the abscess, whose contents will receive an admixture of lymph, till, in consequence of inflammation around the abscess, the vessel ceases to be permeable.

When the above-described changes present themselves in a lymphatic vessel, no doubt can be entertained regarding its inflammation; but we very often meet with lymphatics in a condition presenting many essential similarities with inflammation, and yet, according to our views, not actually inflamed. Thus we often find the lymphatics proceeding from inflamed parenchymatous organs, or from abscesses filled with pus, varicose, dull, and pilous on their inner surface; while, further, we observe that the surrounding cellular tissue presents a vascular and infiltrated appearance, as is very commonly seen in the lymphatic vessels of the hypogastric and lumbar plexuses after delivery. There are, however, absent, on the one hand, the infiltration and swelling, giving rise to the loosening of the coats of the vessel, while, on the other, the injection and infiltration of the retroperitoneal cellular substance investing the lymphatic plexus appear in puerperal cases as an integral part of the peritonitic process. We believe that in these cases the pus is not, or at all events is not always, produced in the lymphatic vessel itself, but is conveyed there from the inflammatory centre or abscess, whether it reach the lymphatic vessels by absorption of the purulent fluid, or has exuded into their cavity; or, finally, whether it has been taken up by lymphatics opening into the abscess;—that the dilatation of the lymphatic vessels arises from the accumulation of pus within them, since its further transmission is impeded by the swelling of the lymphatic glands;—and, finally, that the loss of lustre presented by the inner coat is induced by the loosening and fusing action of the pus.

On the other hand, it is unquestionable that a lymphatic vessel containing pus not unfrequently becomes inflamed, probably in consequence of its coats imbibing pus. The period which such an inflammation occupies is frequently a long one; this is analogous to the singularly long period of incubation, which occurs in the case of poisoned wounds, from the time of the injury to the formation of a decided inflammation of the lymphatic vessels, and to the impunity with which the lymphatic vessels can convey all varieties of ulcerous products and contagious matters, while the glands are highly affected. This indicates that the lymphatic vessels possess a very subordinate sensitiveness to the irritation produced by the contact of heterogeneous matters, especially as compard with the lymphatic glands.

The appearances presented by inflammation of the larger lymphatic vessels, are in accordance with the observations which have been already made; as in phlebitis, *inflammation of the coats of a lymphatic vessel may be the primary phenomenon*, which occasions an anomaly of its contents by exudation into the canal of the vessel, or *inflammation may be excited by the presence of a heterogeneous substance within the lymphatic vessel.*

An infection of the blood by the matter produced in the lymphatic vessel, or absorbed into it from without, and the secondary (metastatic) phenomena consequent on such an infection, are in general rare; the rarity being in proportion to the distance of the process from the central anastomosis of the lymphatics and bloodvessels, and to the number of glands through which the heterogeneous substances contained in the lymphatic vessel have to pass.

Inflammation of the lymphatic vessels is often observed along the course of an inflamed vein; this may sometimes arise through the inflammation of the common cellular bed of the vein and the lymphatic vessel, and may sometimes be dependent on the same cause as the phlebitis, namely, the absorption of heterogeneous matter.

b. Adventitous Products.—These are limited to *Tubercle* and *Cancer*. Each occurs in a special form, as an adventitious mass closing the tube of the vessel, and invariably as a secondary phenomenon. In order that they may occur, there must be an absorption of softened tubercle, or of cancerous blastemata, into the lymphatic vessel. The lymphatic glands act as centres of absorption of the morbid matter, occasioning tuberculosis and cancer of those organs. Tuberculous pus and the cancerous blastema coagulate with the other contents of the lymphatic vessel; the former into a yellow cheesy, the latter in a whitish, more or less brain-like (encephaloid) molecular mass, which finally closes the nodulary dilated lymphatic vessel. Lymphatic vessels plugged with tuberculous matter sometimes present thickened coats and a lardaceous infiltration, doubtless in consequence of having undergone inflammation.

We may often observe both these forms, particularly tuberculosis of the lymphatic vessels, which especially occurs in the lymphatics between the intestinal and mesenteric glands, between the different mesenteric glands, and between the latter and the glands of the lumbar plexus, in tuberculous ulceration of the intestines in tuberculous disease of the mesenteric glands (cavities in the glands), &c.; cancer of the lymphatics especially occurs in cases of medullary cancer.

§ 2. *Anomalies of Calibre.*

A moderate dilatation of one or more lymphatics, which may either be *uniform* or *nodular* (*varicose*), is by no means rare; it may be dependent on pressure or on the impermeability of some of the coils in lymphatic glands. The coats of the vessel are in these cases sometimes relaxed and attenuated, and sometimes thickened. That certain cysts and hydatids (such, for instance, as the structures occurring in the choroid plexus of the lateral ventricles, and formerly regarded as hydatids), consist of varicose lymphatic vessels, as is taught even in the present day, is, in our opinion, by no means proved: but this does not exclude the possibility that lymphatics may sometimes assume a bladder-like dilatation at certain spots, as, for instance, between two pairs of valves, where they may present a constricted appearance. An extraordinary and very rare example of general dilatation of the lymphatics has been recorded by Breschet (Le Syst. Lymph., Paris, 1836), for which he was indebted to Amussat.

Contraction of the lymphatics occurs independently in general or partial atrophy, and arises from the compression exerted by every variety of tumor. It is also manifested in the form of occlusion—obliteration.

B. *The Lymphatic Glands.*

§ 1. *Anomalies of Volume—Hypertrophy—Atrophy.*—The lymphatic glands are *abnormally enlarged* in consequence of various conditions. We have here to consider more fully the anomalies induced in these structures by hypertrophy. This condition consists in an excessive accumulation of parenchyma between the lymphatics interspersed through the gland. In the present deficient state of our knowledge in reference to the structure of the lymphatic glands, and to the signification of their parenchyma, we must include under the above head all enlargements of the lymphatic glands which do not depend upon hyperæmia, inflammation, or any obvious secondary formation; although it cannot be doubted that by such a classification we are compelled to include with hypertrophies many specific alterations of the glandular parenchyma. We are as yet unacquainted with the mode of its origin, as well as with the manner in which the so-called lymphatic diathesis or habitus is induced, and with the exception of the little that is known in reference to hypertrophies generally, we are ignorant of the connection that may exist between the hypertrophied development of the lymphatic glands and the co-existing disturbances of the general organic system. We are inclined, from the little that is known in reference to the subject, to regard hypertrophy of the lymphatic glands as a secondary, symptomatic phenomenon, and not as a primary and substantive anomaly.

Hypertrophy of the lymphatic glands is most common in childhood, and until the full development of puberty; although it is not unfrequently exhibited after that period, and even in mature life. The lymphatic glands of the abdomen, of the mesentery, and of the lumbar plexus, are the most frequent seats of hypertrophy. This condition of the lymphatic glands of young persons is very commonly associated with a hypertrophied development of other blood-forming glands, as, for instance, the thyroid gland, and more especially a highly developed spleen, obstructed involution of the thymus, a hypertrophied development of the follicular apparatus of the intestines, and hypertrophy of the nervous centres. Such hypertrophies either affect the whole system generally, or one portion especially, as, for instance, the glands of the abdomen.

Abnormal smallness is the result of *atrophy*. The lymphatic glands disappear in advanced age with the symptoms of general tabes, until their presence can scarcely be detected. This atrophy either affects the gland uniformly at all points, or preponderates at certain spots, where the parenchyma is entirely destroyed, leaving nothing but a white, soft, cellular, shrivelled tissue. The parenchyma which remains in diminished quantity, either at the periphery or in the centre, and presents either the appearance of a capsule or a central accumulation, very commonly acquires a dull, rusty-brown color. It is not improbable that the gland may first be reduced to the condition of a simple lymphatic nodule, as in its primary fœtal state, and that the lymphatic vessels may also subsequently become atrophied together with the capillaries.

Moreover lymphatic glands may become secondarily atrophied in consequence of inflammation, and more especially of specific inflammatory processes. A marked degree of atrophy of this kind not unfrequently affects the mesenteric glands in consequence of typhous infiltration. The parenchyma of the gland is in this case absorbed, together with the product of the process. The involution of the typhoid mesenteric glands, which ultimately degenerates into tabes, has been fully considered under intestinal typhus. A similar atrophy occurs in adventitious products, partly in consequence of mechanical pressure and partly from inflammation in the neighborhood of the adventitious structure.

§ 2. *Anomalies of Texture.*

a. Inflammation.—Inflammation of the Lymphatic Glands (*Lymphadenitis*) especially when it depends on the absorption from within of heterogeneous substances into the lymphatic vessels, is of frequent occurrence in comparison with lymphangioitis. These substances may either be inflammatory products, or different contagious or deleterious matters, originating in inflammation; a distinction on which is based the difference existing between consensual inflammation of the lumbar glands and syphilitic bubo in chancre. Substantive inflammation of a group of lymphatic glands, or of a single gland, is rarely independent of the abovenamed modes of origin, although some exceptions present themselves in the case of various specific inflammations, more especially when of a typhous character.

The lymphatic glands far exceed the lymphatics in the readiness with which they absorb heterogeneous substances, in consequence, perhaps, of the vascularity of the former, and of the manner in which their whole structure is permeated by the bloodvessels and the lymphatics; and, lastly, in consequence of the transference of matter occurring between these two systems of vessels.

A lymphatic gland presents the following alterations when in a condition of *recent acute inflammation.*

The gland is injected, and presents various shades of redness; it is swollen, relaxed, soft, and lacerable; its tissue is uniformly permeated by a serous, fibrinous, purulent exudation, or is suffused at individual points with large quantities of the same fluid. The original redness is thus variously altered, and in some cases the gland even appears as if its reddened structure were interspersed with different spots and stripes, in consequence of the absorption at individual centres of a coagulable exudation. Considerable hemorrhage is sometimes observed to occur in the glands during the continuance of the inflammatory process, and, in that case, they are found to present variously sized centres filled with blood in different stages of coagulation and discoloration. The cellular bed of the gland participates in the process when it exhibits any considerable degree of intensity; and the former is then injected, reddened, and infiltrated, that is to say, inflamed, and the gland becomes adherent to it. It is, moreover, not unfrequently ecchymosed by small extravasations of blood.

This inflammation *terminates* in various modes, very commonly in resolution; even large quantities of solidified exudation may be readily absorbed. It not unfrequently terminates in *induration*, in which case the copiously deposited exudation becomes converted into a fibroid callus, whilst the glandular substance is either partially or wholly atrophied. Finally, intense inflammations may result in *suppuration of the gland—abscess—phthisis of the lymphatic gland.*

Chronic Lymphadenitis is in reality a protracted inflammation of moderate intensity, with occasional acute relapses, in which the swelling of the lymphatic gland is commonly not so considerable as might be expected, in consequence of the resorption of the exudation which occurs during the remissions. It terminates in resolution through the suspension of the processes by which it had been maintained, and not unfrequently in induration with atrophy of the glandular parenchyma. The cellular bed in which the gland lies is more or less affected by the induration.

Inflammation of a gland, or of a group of glands, may readily give rise to inflammation of the adjacent glands, by the transmission of the products taken up by resorption; in this case, however, even if there is suppuration of the glands, it is seldom that the blood becomes so affected as to lead to a fatal termination.

As inflammations with a special product we must especially notice *inflammation with tuberculous exudation—typhous inflammation.* We shall treat of the former in our remarks on adventitious products; and the latter is fully described in our observations on Intestinal Typhus. We must, however, here especially notice the following points:

Typhous inflammation of the lymphatic glands occurs amongst us [at Vienna] as inflammation of the mesenteric glands, associated with the typhous process on the mucous membrane of the ileum, or, strictly speaking, on its follicular apparatus. It forms an integral part of ileotyphus. It particularly attacks the chain of lymphatic glands which corresponds to the affected part of the intestine, and extends from the lowest portion of the ileum till it implicates the glands of the lumbar plexus. We have here the very important question to decide—Is the inflammation of the mesenteric glands, which occurs in ileo-typhus, and is characterized by its peculiar product, an affection dependent on and secondary to that of the intestinal follicles, like the inflammation of the lymphatic glands in chancre and in certain diseases of the scalp, or is it an independent and substantive localization of the general process?

The former view would seem to derive confirmation from this circumstance alone, that the typhous disease of the mesenteric glands is, at first, subordinate in intensity to the degree of disease affecting the follicular apparatus, and that the typhous metamorphosis of the mesenteric glands only in exceptional cases precedes that of the typhous structure in the intestine. The latter view of the question is, on the other hand, supported by several circumstances.

a. The typhous matter cannot be traced in the lymphatics during its transference from the intestine to the lymphatic glands, nor from its coagulability does it admit of such a transference in the more intense degrees of typhus; but, notwithstanding this, the mesenteric glands are already infiltrated before the loosening and softening of the typhous placque in the intestine.

b. In anomalous forms of typhus, the mesenteric glands are obviously in a typhous condition (together with the spleen), although indeed in an inconsiderable degree, while the contiguous intestinal mucous membrane is entirely exempt.

c. In many of the more rare cases, the local process extends with excessive intensity to the mesenteric glands, without in any way affecting the mucous membrane of the intestine.

d. In bronchial typhus, the mucous membrane of the bronchi, like that of the intestines, is entirely exempt from the production of the typhous structures; for, while the typhous process is here limited to the stage of congestion and typhous catarrh, it is exhibited, in a very highly developed form, in the bronchial glands.

e. In the Oriental Plague, even the lymphatic glands, which are in no way connected with the mucous membranes (as, for instance, with that of the intestines), are diseased.

We are led to conclude, from the above considerations, that the disease of the mesenteric glands in ileo-typhus is a substantive affection, on the one hand from the near relation exhibited by our own typhus to the lymphatic system—a relation which is so obviously manifested in the highly developed form of plague; and on the other, from the affinity between the true follicular apparatus of the intestines and the lymphatic system.

Next in order to typhous inflammation of the lymphatic glands, we proceed to consider—

b. Acute Swellings of the Lymphatic Glands.—These are morbid conditions of the lymphatic glands (more especially those of the mesentery), which occur in the form of acute intumescence, associated with some degree of vascularity, and with loosening of the tissue. Although these conditions very probably differ considerably in their inner character, we are as yet but very imperfectly acquainted with their nature; and hence we are able to do little more than refer to them under the above designations, which are borrowed from the most striking appearances which they present. However nearly they may seem, at first sight, to be allied to hypertrophies, for which they are very generally mistaken, they are yet very different. Inasmuch as they are developed in an acute form, and always occur simultaneously with acute diseases, which are essentially manifested as dyscrasiæ and neuroses, we think they must be regarded as the localization of a general process of disease, and that the structure on which the increased volume of the gland depends is of a specific character. This circumstance forms the basis of the indication correctly deduced from these appearances, that they cannot be regarded as pre-existing developments connected with a chronic anomaly of the general condition of the organism, but must rather be considered to refer to an acute dyscrasia. On this account we have noticed them next in order to typhous inflammations of the lymphatic glands. They commonly, or at all events most strikingly, affect the mesenteric glands; and here, as in typhus the follicular apparatus of the intestine is almost invariably diseased in a similar manner.

If we pass over the swelling of the glands observed in Asiatic cholera, and which is explained provisionally at all events, by the tumultuous hyperæmia and formation of products in the whole intestinal apparatus, we may reckon the above described forms of glandular intumescence as characteristic of acute exanthemata, such as scarlet fever and variola, and of acute convulsions, such as epilepsy, tetanus, and trismus, both in children and adults. They are further observed in numerous dyscrasic, febrile, and more or less genuine typhoid conditions, and are manifested during life by a complication of symptoms, and after death by a combination of anatomical alterations.

We do not think that we are in error in reference to the above observations, although, as is obvious, everything relating to this subject is still merely conjectural.

c. Adventitious Products.—The most frequent and important of these are tubercle and cancer.

1. *Formation of Cysts.*—This is of very rare occurrence, more especially when we except the formation of cysts in the lymphatic glands in association with cancer. Varicosities of the lymphatics in the glands must not be confounded with cysts, as was formerly done, nor must they be mistaken for the apertures which are occasionally observed in the stroma of the impoverished parenchyma of atrophied glands. There is an old preparation in our Museum, in which the glands of the lumbar plexus have degenerated into tumors of the size of a pigeon's or hen's egg, and which appear like a convolution of somewhat large sacs, intermixed together, and having comparatively thick walls. It is impossible to form a correct idea of the nature of these cyst-formations.

Sacs having purulent, cheesy, and greasy contents, or which are filled with a chalky paste, and are occasionally incrusted with mortar-like walls, are obsolete abscesses—tuberculous caverns.

2. *Black Pigment.*—Large accumulations of this substance frequently occur, as is well known, in the bronchial glands. It is also occasionally found, in smaller quantities, in the mesenteric glands, and even in other lymphatic glands. The bronchial glands are often so swollen with this substance, that they appear like considerable, inky, tough tumors. It is the residuum of the hæmatin, which has been deposited in the course of

hyperæmia and inflammation of the glands. The blackness of the bronchial glands, is associated with the well-known accumulation of pigment in the parenchyma of the lungs, and it is supposed that a part of the pigment here formed is absorbed by the lymphatics, and deposited within the bronchial glands. The black color of the mesenteric glands coincides with the discoloration of the mucous membrane round the apices, and supposed openings of the excretory ducts of the solitary and agminated gland-capsules, and with the blackened appearance of the intestinal villi. It is particularly marked after typhous congestion and inflammation.

3. *Tuberculosis.*—*Tuberculosis of the lymphatic glands* is next to that of the lungs and the intestinal canal, the most frequent form of tuberculous disease, and more especially affects some portions of the lymphatic system, as the bronchial and mesenteric glands, and those of the neck and lumbar region. Children are peculiarly liable to this affection.

Experience yields the following facts in reference to the form in which tubercle occurs in the lymphatic glands.

a. Sometimes we find scattered through the lymphatic glands, ramifying or grouped together, or confluent, roundish, grayish, semi-transparent or turbid, opaque masses of the size of a poppy or millet-seed. Some of these masses present a yellow color and a relaxed texture. There is no doubt that this is the same structure to which, in other places, and especially in the lungs, we apply the name of gray, crude tuberculous granulations.

b. In other cases, and more frequently, the lymphatic glands are plugged up with large roundish or irregular ramifying, greenish-yellow, yellow lardaceo-caseous, hard, although brittle, cleft and gaping, caseo-purulent, fusing masses, into which the lymphatic glands appear to have actually degenerated: the glands, in this case, form tumors, which vary from the size of a hazel-nut to that of a hen's egg, or may be even larger. The glandular substance sometimes, and most commonly, surrounds the adventitious matter, forming, as it were a capsule round it; while sometimes it traverses the latter in the form of stripes, in considerable accumulations. In each form it sometimes appears vascularized, relaxed, and here and there penetrated by one or other of the above-named granulations or smaller yellow, caseous masses, and sometimes it is thickened, indurated, or atrophied. We observe swollen lymphatic glands, arranged like a knotted rope along the jugular veins in the neck, in the mesentery, and along the trunks of the vessels on the lumbar vertebræ, crowded over one another into nodular heaps around the cysterna lumbalis, the head of the pancreas, the biliary ducts, the bronchi, &c.

The question now arises, whether these two forms are only various stages or different degrees of development of one and the same adventitious product. We are convinced that, in the lymphatic glands, in the same manner as in other parenchymatous structures—as, for instance, the lungs—the gray tuberculous granulations may be so accumulated, that they at length run together into considerable and apparently homogeneous masses, and, as they become yellow, constitute the cheesy nodules which are described under 2. But we do not believe that this view holds good for all cases; we are rather of opinion that the tubercle of the second form is the (tuberculous) product of an inflammation of the lymphatic glands. In favor of this view we may notice:

1. The analogy of the adventitious mass with the tuberculous exudation in other tissues.

2. The coincidence of this tuberculosis with tuberculous inflammation—as, for, instance, tuberculosis of the mesenteric glands with tuberculous inflammation of the follicular apparatus of the intestines; tuberculosis of the bronchial glands, with a similar disease of the bronchial mucous membrane, with pneumonic pulmonary tubercle, &c.

3. The homogeneous character of the adventitious product throughout its mass, as well as—

4. In a large number of lymphatic glands, in addition to the absence of tubercle, in the form of gray granulations, or at most to its presence in very inconsiderable quantity.

5. Finally, the painfulness of the diseased gland, at all events at the beginning, and the accompanying fever.

Each of these forms of tubercle of the lymphatic glands not unfrequently undergoes the softening metamorphosis, giving rise to *tuberculous caverns and ulcers of the lymphatic glands, or tuberculous phthisis of the lymphatic glands.* The caverns, according to their position, open into the serous cavities, into the intestinal canal, or into the bronchi, in cases of mesenteric or bronchial glandular disease, and very often externally after suppuration of the general investments, as in tuberculosis of the cervical glands.

Sometimes, and especially in the bronchial and mesenteric glands, the tubercle undergoes the *process of cretefaction.* Its place in the gland is then occupied by a roundish, solid, or partially hollow, always uneven, nodular, tuberose, often ramifying concretion, corresponding to it in size, form, and arrangement; this concretion is often enclosed as in a capsule, and traversed by atrophied, callous, indurated parenchyma, or by a portion of gland still capable of performing its normal functions.

Tuberculosis of the lymphatic glands may be *primary,* in which case it is either confined to a certain section of the system—as, for instance, the mesenteric glands, or is diffused over nearly the whole system—as, for instance, the glands of the body generally. In the first case it is not unfrequently perfectly *isolated* and *independent,* although it is more commonly associated with tuberculosis of the organs in the immediate vicinity of the diseased glands; thus, for instance, we have tuberculosis of the mesenteric glands with tuberculosis of the small intestine, and tuberculosis of the bronchial glands with tuberculosis of the bronchial mucous membrane and the lungs. We regard this combination in most cases, and especially in intense cases, *as an original one,*—that is to say, we believe that the two structures, as, for instance, the intestinal mucous membrane and the mesenteric glands, are simultaneously affected.

Or the tuberculosis of the lymphatic glands may be *secondary,* and *dependent* on tuberculosis of some other organ. The above-named combinations may serve as illustrations; thus, for instance, tuberculosis of the mesenteric glands is often associated with tubercle, and especially with ulcerating tubercle of the intestinal mucous membrane. In such cases we see the lymphatics proceeding from the intestine, and especially from the seat of the ulcer, filled with yellow cheesy tuberculous matter.

Tuberculosis of the lymphatic glands frequently acts as a starting-point for other tuberculoses, and especially for those of the serous membranes.

With the exception of pulmonary tuberculosis, when limited to the apices of the lungs, no tuberculosis becomes healed so frequently as the form we are now considering, when limited to one of the smaller groups of glands. The cure is effected by the suppuration (phthisis) of the gland, and the discharge of the pus externally (as, for instance, in the neck), or by cretefaction of the tubercle (as, for instance, in the bronchial and mesenteric glands).

When, however, tuberculosis of the lymphatic glands is very widely diffused, it may prove fatal, either of itself, or in connection with other pre-existing, simultaneous, or consecutive tuberculoses through tabes, before undergoing the above-named metamorphoses.

The special *seat* of tuberculosis of the lymphatic glands—regarding it as a secretion from the capillary vessels—is the parenchymatous structure; it is, however, not improbable that the second form of tubercle is exuded into the interior of the lymphatics, and effects their occlusion. The same may happen when tuberculous masses are absorbed by the lymphatics, and transferred to the gland.

4. *Cancer* frequently occurs in the lymphatic glands, sometimes as a primary, but more commonly as a secondary formation. The *medullary* is the ordinary variety, either in its genuine white form, or in association with melanosis; or sometimes combined with areolar cancer, or with cysts.

In the *primary* form, it especially attacks the glands of the lumbar plexus, and those in the mediastina; in both these positions it forms considerable tuberous growths, which, in the former, are known as retroperitoneal masses (Lobstein). Next in order of frequency, it occurs in the axillary, lumbar, and cervical glands. It is extremely probable that many of the cancerous structures imbedded in the cellular substance, and in which no starting-point from any other definite organ can be detected, on account of the integrity of the surrounding parts, originated in one or more lymphatic glands.

It appears in a secondary form when it does not develope itself in the lymphatic glands of a parenchymatous organ until that structure has already been affected with cancer. This cancer is sometimes *very rapidly* developed. In these cases the cancerous product is always distributed over a large, generally over the greatest, part of the system; it is also usually combined with acute cancer in other organs, especially the lungs and spleen.

The *seat* of the cancer is the parenchyma of the gland; but, at all events, in cases of secondary cancer, where the disease has been occasioned by the absorption of cancerous matter into the lymphatics, the cancer may also be seated in the lymphatic vessels of the gland.

5. *Entozoa.*—We must here notice the animal found by Treutler in the bronchial glands, and named by Rudolphi, *Filaria hominis bronchialis.*

§ 3. *Anomalies of Contents.*

These have been already noticed in the preceding pages. We may add that, once in a medico-legal examination of the body of a man, aged about 35 years, the cause of whose death was unknown, but who probably died in convulsions, and in whose intestinal canal there had been a considerable development of gas, we found several of the mesenteric glands and of the lymphatics proceeding from the intestine in a state of emphysematous inflation, which we were the more inclined to attribute to the absorption of the intestinal gas, seeing that the character and appearance of the body generally were opposed to the view that there had been a development of gas as a consequence of putrefaction.

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